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SIDEROSIS BULBI.

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CLEVELAND, OHIO.

A patient without history of injury presented a perforation of the iris, traumatic cataract, exudative pupillary membrane and beginning siderosis. Evidence of a corneal scar was brought out only by slit lamp microscopy, and X-ray examination proved negative. Removal of the eye showed that siderosis extended to the iris and lens but no foreign body was found. The injury was probably received when hammering on enameled steel. Reported from Lakeside Hospital, paper read before Ophthalmological and Oto-Laryngological Society of Cleveland, November, 1925.

The following case was thought of sufficient interest to report, first, because it is of unusual interest, in that the patient gave no definite history of an injury of his eye. Second, that the entrance wound in the cornea could not be disclosed by our ordinary methods of examinations. Third, because the Ohio Industrial Commission disputed the claim of the patient for a period of two years. This was due to the fact that the several ophthalmologists who examined the case could not agree that the eye condition was one directly due to trauma. Fourth, that the slit lamp microscope was the deciding factor in bringing to view the site of injury in the cornea and clinched the diagnosis, so that the patient was immediately awarded compensation for the total loss of the eye by the Ohio State Industrial Commission.

My diagnosis at the time the patient consulted me October 23, 1924 was as follows:

Right Eye:

(1) Exudative pupillary membrane.
(2) Cataract, traumatic.
(3) Perforating wound of iris.
(4) Beginning siderosis.

On October 23, 1924, Mr. G. H., age 42, employed as an enamel burner in a large manufacturing plant in this city, came in with the following:

Complaint: loss of vision in right eye. Past history and family history negative. Present illness. Onset in May, 1923, when he noticed that his right eye was painful and red and light bothered him. Consulted the industrial surgeon where he was employed and was treated for a few days without beneficial results. The plant surgeon referred patient to an ophthalmologist who had the case under observation and treatment for six weeks. A diagnosis at that time was made of acute iritis of unknown origin, after all laboratory tests had proven negative. The patient was discharged and returned to his employment. However, the patient states that he is unable to carry on his work as formerly because of the reduced vision in right eye, which he is positive was normal before the onset of his trouble.

Examination showed the following: Left Eye: Normal in every respect. Right Eye: V. O. D. Light perception only. Projection normal, muscle movements normal. Slight palpebral and ocular conjunctival injection. Externally the cornea was clear. The pupil was regular, with no adhesions, 4 mm. in diameter, and reacted to light and convergence. Anterior chamber slightly more shallow than left. Tension 15 mm. Hg.

Examination under mydriatic. In the entire pupillary space was to be seen an irregular projecting greyish thickened membrane, with minute lemon yellow spots in its substance and on the pointed ends of the same. Also on the posterior layer of the cornea are seen several of these same spots, which

measured about 1 mm. in diameter, and are situated above and below peripherally. A direct view of the lens was not possible due to the membrane, but several striae are seen posterior to the membrane that appeared to be in the anterior portion of the capsule, and in lens proper, giving the impression of a traumatic cataract. The fundus was not discernible, only the glow being seen.

In the iris surrounding the pupillary border there is a zone which extends 3mm. peripherally, which has a distinct dull greenish brown discoloration and is unlike the color of left iris. It may be of interest to note, that in the iris in the region of 4:30 o'clock, there is a small clean cut opening measuring 2.5 mm. by 1 mm. Transillumination disclosed this aperture very distinctly.

The discovery of this iris opening brought up the question as to whether we were dealing with a congenital defect or the result of a trauma brought on by some sharp foreign body. The patient gave no history of an injury, but stated that he occasionally hammered on enameled substance and steel, and at one of these times a piece of sharp enamel or a piece of scale of steel may have penetrated his eye. However, examination failed to disclose any opacity of the cornea. A foreign body may have entered the iris tangentially somewhat apart from the scleral corneal margin. To eliminate a possible foreign body an X-ray of the orbit was made by Drs. Hill and Thomas, but the findings were negative to metallic foreign body. If an enamel piece were present the X-ray would not of course show it.

In as much as there were conflicting statements as to the causation of the patient's ocular condition, it was felt that in justice to the man, every means known in the field of ophthalmology should be resorted to in order to establish the etiology.

At the time the patient consulted me, he was advised to have his case reopened before the Ohio Industrial Commission, as I was positive that he had a just claim. Thru the patient's attorney the case was reopened and my report was submitted and again the claim was disallowed.

When Professor Leonard Koeppe of the University of Halle, Germany, was here last June giving a series of lectures on the "Slit Lamp Microscopy of the Living Eye" I consulted with him on Mr. G. H.'s case, and he consented to examine the case with me, using the slit lamp microscope.

The following is the report of the ophthalmologic slit lamp examination of G. H. made on June 27, and June 29, 1925 by Professor Leonard Koeppe.

Under mydriatic: Right Eye: Examination discloses a perforating scar in the cornea in the region of 3:45 o'clock which is triangular and pigmented with iron pigment. The wound is 5 mm. in diameter.

The largest side of this triangle is directed horizontally and the other sides are directed downward. The hole is extremely fine and transparent and pigmented with green pigment, consisting of very fine pigment particles in the depth of the cornea. Corresponding to the nasal side of the triangle, one can see an oblique fold 3 mm. long in Descemet's membrane which is covered with endothelium and formation of a new Descemet's substance in the form of a long strand. This is certainly a healed extreme scar of a foreign body. Also the whole cornea shows a yellowish color of the whole lymph-slit system and quite dense pigmentation of the posterior surface. The cornea is in a state of typical general siderosis. The anterior chamber is without changes.

Corresponding to the corneal entrance scar, but located about 5 mm. deeper, we find in the iris at a distance of 2 mm. from the periphery of iris a long elliptical perforation in the iris tissue. In the opening in iris, a few ruptured fibers of the ligamentum suspensorium are visible. Also there are ruptured iris fibers in all parts of the perforation. The other tissue of the iris is in a normal state, altho certainly a little more greenly colored in comparison with the other eye. Most certainly the iris is affected with

siderosis, caused by an iron foreign body situated somewhere in the eye. The iris pigment border is normal.

In the pupil is to be seen a shrinking traumatic hypermature cataract. This cataract shows in the white degenerated capsule many greenish yellowish colored pigmented bodies partly in form of spots, and more diffusely. Here in the lens capsule is also a siderosis. The complicated cataract and the siderosis are the result of the injury of the lens by the foreign body. Therefore, I am of the opinion, also because of the exudate located on the capsule, that the injury was the original cause of the cataract.

No foreign body was noted with the slit lamp and the X-ray was negative, but we have very often a negative X-ray finding when the foreign body is very small. Because of the slit lamp findings there is no doubt that the present pathologic state of the eye was caused by a piece of iron metal penetrating the eye. Small particles of iron, as for instance rust on a piece of iron penetrating the eye, but not remaining itself, will often give the same picture.

Enucleation was advised, consented to and performed December eight, 1925. The patient had an uneventful recovery.

The following is the pathologic report of the eye as submitted by Dr. H. S. Thatcher, pathologist of Lakeside Hospital.

Case of G. H.

"I have made numerous sections of the eye and found the area where the penetrating wound was present. I have made sections thru practically all of the eye, and find the condition described."

HISTOLOGIC.

The regular contour of the corneal tissue is destroyed. The epithelial cells are thickened here, and irregularly arranged. There is also a break in Bowman's membrane. The thickest connective tissue layer has been disturbed, and there is a bulbous change. The new connective tissue is arranged irregularly and compact. This is more pronounced in the anterior portion, but there is a region of fibrosis near the iris. The iris is also more fibrous than normal. There is considerable rusty brown discoloration of the iris, especially on the outer edge. The lens is slightly discolored yellowish brown in the external layers.

DIAGNOSIS.

Siderosis bulbi; localized scar tissue in the cornea and iris.

This case is no doubt only one of the many where the slit lamp microscope has proved of value in clearing up the diagnosis. My limited experience with this comparatively new ophthalmologic instrument has convinced me that a wider and more general use of the instrument would uncover the etiology of a great number of unsolved eye problems. Also in medicolegal cases the slit lamp microscope is destined to play a very important role.

Before closing the report of this interesting, instructive and unusual case, we feel that a few pertinent remarks pertaining to the manner in which it was handled will not be amiss. Certainly an injustice to this man was done in forcing him to fight for his rights over a period of two years. Besides losing his eye, the patient was placed under a heavy medical and legal expense, with which he should not have been forced to burden himself. The responsibility for this rank injustice should be placed squarely on the shoulders of the employer. True it is that there was an element of doubt in the case, but it was quite insignificant. There is something radically wrong somewhere in a State Compensation Act that will tolerate such a condition to exist.

The proper course that should have been followed in this case and any other of like nature, would have been the appointment by the proper State Industrial Authorities of a board of three qualified and recognized ophthalmologists to pass on the merits of the case. The ultimate result of such a procedure would have been the early disposition of the case, thus eliminating the necessity of litigation and the consequent loss of money and time to the patient.

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HEMANGIOMA OF THE CHOROID.

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A brief review is given of the twenty-five cases previously reported. The details of the authors' case, as seen clinically and demonstrated after removal, are described.

Hemangioma of the choroid is a rare affection of the middle coat of the eye in which the normal choroid tissue is replaced to a greater or less extent by a small cavernous hemangioma in the vicinity of the nervehead, often producing an intraocular tumor, lenticular in shape and of considerable size, and usually, ultimately ending in degenerative changes in the retina, and secondary glaucoma with all its attendant changes.

The apparent rarity of this tumor of the choroid is attested by the fact that a careful search of ophthalmic literature reveals but 25 previously reported cases that have been confirmed by pathologic examination. In addition to these certainly diagnosed cases, the literature contains reports of 5 cases in which hemangioma of the choroid was diagnosed clinically, but not confirmed by biopsy. As the diagnosis of choroidal hemangioma offers many difficulties, and as most of the pathologically demonstrated cases, including our own, were not diagnosed ophthalmoscopically, these five clinical cases (those of Fehr, 1905, second case; Galezowski, 1898; Deyl, 1899; Henderson, 1920; Strader, 1924) are not included in the statistical review of the subject.

The earliest reported case appears to be that of Leber, 1868, and the latest case that of Sobhy Bey, 1923. These cases have been culled from the literature as follows: German 16, English 3, American 2, French 1, Italian 1, Norwegian 1, Egyptian 1.

The diseased condition is probably more common than the reported cases indicate. If all eyes removed for new growths, advanced glaucomatous condition, etc., were sectioned, many cases would probably be brought to light; in fact, that is the manner in which most of the 26 known cases were discovered.

The cause of hemangioma of the choroid is probably the same as that of hemangioma elsewhere in the body. So far as can be learned from the literature the cases appear to have been congenital. In the 25 reported cases 12 (nearly 50%) had hemangioma or vascular nevi elsewhere on the body ranging from a small one, only 3 mm. in diameter, over the head of the sternum, to extensive ones involving one side of the face and in one instance both sides, have been present. Love, 1914, gives an interesting account of the possible etiology of angiomas.

The age incidence for the previously reported cases and the present one is

birth—10 years	3
11—20 years	11
21—30 years	3
31—40 years	3
41—50 years	3*
51—60 years	1*
61—70 years	2*
71 and more years	0
Age not stated by author, other than adult	2

As to sex, 14 cases occurred in males and 11 in females; in one case the sex not stated.

In ten of the cases (about 40%) the right eye was involved, in 15 (about 60%) the left eye, and in one instance both eyes, the left only being enucleated. The greater preponderance of cases in which the lesion occurred in the left eye is probably due to the small series available for statistical study rather than to any marked predilection for that eye for choroidal hemangiomas.

Five patients (cases of Lawford, Michail, Panas and Remy, Reis and Wagenmann) had injuries to the affected eyes before consulting ophthalmologists. Three of these injuries were blows, one a foreign body, and in one wood tar was sprinkled in the eye. The injuries themselves probably had nothing to do with causing the onset of angioma, but were contributory in bringing about the end results, such as glaucoma.

*Fehr's first case observed first in the 5th decade, and removed in the 7th decade.

The tumors as seen in the enucleated eyes are essentially lenticular in shape, following for the most part the natural curves of the eye coats and located about the nervehead. They have been described as the size of a "pea," the size of the crystalline lens, as having dimensions of three to four papilla widths. Those actually measured range from 2 to 17 mm. in diameter and in thickness from 1 to 9 mm. The tumors show for most part a simple cavernous angiomatous structure, which replaces the normal choroid. Some authors have described their cases as angiosarcomas, but in the cases reviewed the sarcoma element seems negligible; and the impression is that sarcoma being a well recognized intraocular tumor and frequently diagnosed ophthalmoscopically, a few early authors were led into seeing a mixed tumor instead of a simple angioma.

The position of the growths appears to be always near or around the nervehead, as in the present instance. The larger tumors extend from the nervehead to the ciliary body. There appears to be no special quadrant in which the tumor mass is found most frequently.

Fibroid changes frequently take place in the retinal surface of the angioma often leading to bone formation. Of the 25 enucleated eyes, 15 (60%) showed a layer of bone along the retinal surface, ranging in thickness from 0.25 to 2.00 mm. Bone formation was not found in our case. Paton and Collins express the view that fibrous tissue on the inner surface of the growth results from chronic lymph stasis, and forms the basis on which the bone developes.

Twenty-two of the 25 reported cases developed glaucoma; that is, 88% terminated in that condition. Glaucoma is probably the usual end result of hemangioma of the choroid. Several eyes have been removed before the stage of glaucoma under the mistaken impression that the growth seen ophthalmoscopically was a glioma or sarcoma, as in our own case. Of the 22 eyes which showed a glaucomatous condition, 14 appear to have been removed for that condition alone, and at the time of removal there was no suspicion of any tumor in the eye. The presence of the new growth frequently causes a detachment of the retina, a condition which has been noted at least 13 times, as well as in our case, 54% of the cases. One of the clinical diagnosed cases, that of Galezowski, showed the condition of buphthalmus in both eyes.

The subjective symptoms due to hemangioma of the choroid vary from what patients say is a gradual to a rapid diminution or loss of vision to all the subjective symptoms of glaucoma. Apparently the diminished vision is seldom noticed until the patient passes the first decade. Probably some patients have small angiomas and pass thru life w i t h o u t their causing noticed symptoms. Three authors, Fehr, Reis and Salus, record visual fields which showed contraction, as did our own case.

When the media are clear, the objective symptoms are the presence of a small tumor in the fundus of the eye and evidence of a detached retina. The detachment of the retina may be partial or complete. In the 25 reported cases detachment of the retina was noted in about half of them, 11 cases. The tumor seen ophthalmoscopically is usually small and flattened and is described as grayish white, bluish white, greenish white, bluish green and bright yellow, never dark like a pigmented sarcoma, and never showing signs of inflammatory reaction. In our own case the tumor mass appeared bluish white about the size and shape of a split pea. In only 9 of the 25 reported cases was a tumor mass seen, including our own case, making a percentage of 34. In 6 of the cases reported a detached retina was seen along with the tumor.

The clinical diagnosis of hemangioma of the choroid has very rarely been made. It apparently was made in only one of the 25 cases proved by biopsy, that of Paton and Collins. Five authors, Fehr, Galezowski, Deyl, Henderson, and Strader, have reported

hemangioma on clinical and ophthalmoscopic grounds only.

The points on which a clinical diagnosis may be based are (1) the light color of the tumor, that is absence of pigmentation, (2) absence of inflammatory change about the tumor, (3) its almost imperceptible rate of growth, and (4) according to Salus, an enormous widening of the choroidal vessels

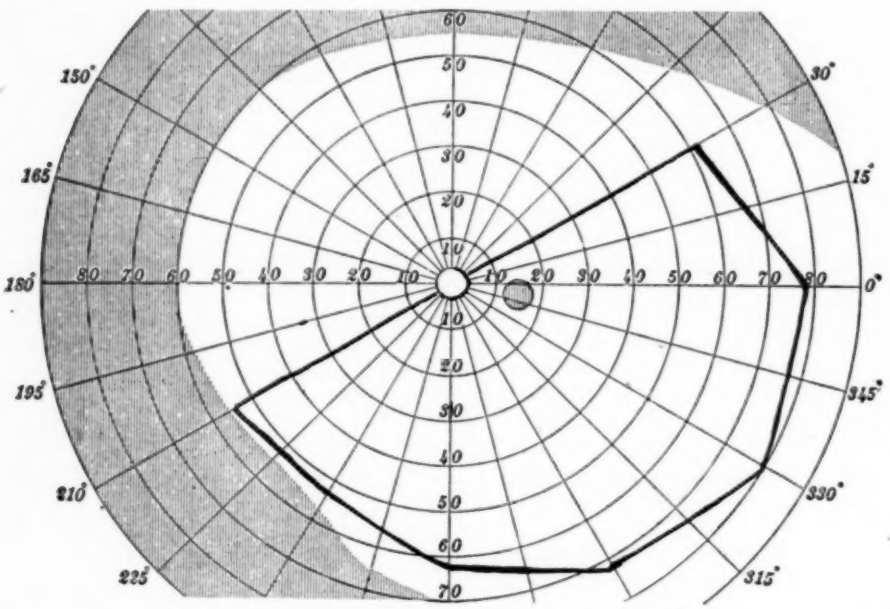

Fig. 1.—Visual fields Sept. 8, 1924, Morton perimeter.

in the peripheral parts of the fundus. In the late stage, when glacomatous changes set in and the media are not clear, it is impossible to make a diagnosis. Presence of an angioma about the face might help one to suspect the condition.

If one assumes that hemangioma of the choroid is congenital, as appears to be the case with hemangiomas of the skin, the condition may persist without inconvenience to the patient through a rather long life time, two cases being reported in the seventh decade. Fehr's first case diagnosed ophthalmoscopically and clinically had been under observation by him for 20 years. During these years vision became diminished and tension slightly increased, and finally secondary glaucoma supervened and the eye had to be removed. Most cases have come under observation when secondary changes were present, or if a tumor has been observed thru clear media, the eye has been removed without waiting to follow the course of the disease.

The prognosis is grave as to sight in the affected eye and saving of the eyeball. Secondary glaucoma has developed in practically all cases in which the diseased eye has been left in. Unlike sarcoma and glioma, hemangioma does not produce metastases and thus endanger life.

Treatment consists in removal of the diseased eye. As long as the eye does not show evidence of glaucoma or other changes, and one is sure of his diagnosis, it would seem well to leave the eye in place as long as possible, as was done by Fehr.

CASE REPORT.

Mrs. B., age 49, first seen in consultation September 6, 1924.

Past History:—Twelve years ago she noticed a beginning dimness of vision in the right eye, at times experienced "maddening pain" thru, right temple. For the past two years she had been under care of an ophthal-

mologist who had "treated" her for "cataract."

Present Examination: — Eyes appeared normal externally, no injection of conjunctivas, corneas clear, pupils reacting to light. No angiomatous lesions were discernible on face or elsewhere.

LEFT EYE, essentially normal, vision 20/20 with a +0.50 diopter sphere. Ophthalmoscopic examination showed a normal fundus.

RIGHT EYE, media clear, vision for lower half of objects. Visual field with Morton perimeter showed absence of vision above the 30° diameter on the temporal side, and above the 180° diameter on the nasal side. (See figure 1.) Ophthalmoscopic examination revealed a bluish white bulging without pigment or signs of inflammatory reaction. It was seen in the lower nasal quadrant, about the size and shape of a split pea. No billowing or waving motions were seen on movement of the eye. Tension by palpation appeared normal. A tentative diagnosis of detached retina due to sarcoma of choroid was made and patient was asked to return in a month.

On October 4, 1924, patient returned. Tension 35 (McLean tonometer), normal. Visual field more contracted in lower temporal and in nasal quadrants. (Fig. 2.) No pain in the eye itself and only occasionally in right temple. Ophthalmoscopic examination showed essentially the same picture as seen a month ago. Enucleation was advised. After consultation with an ophthalmologist in Chicago, who concurred in diagnosis of sarcoma, the eye was removed. Patient made usual uneventful recovery. The eye was preserved in formaldehyd solution and sectioned by Dr. Mary S. Knight, of the Mayo Clinic, to whom thanks are due. Half of the eye and some slides were returned, and on them the following pathologic report is based.

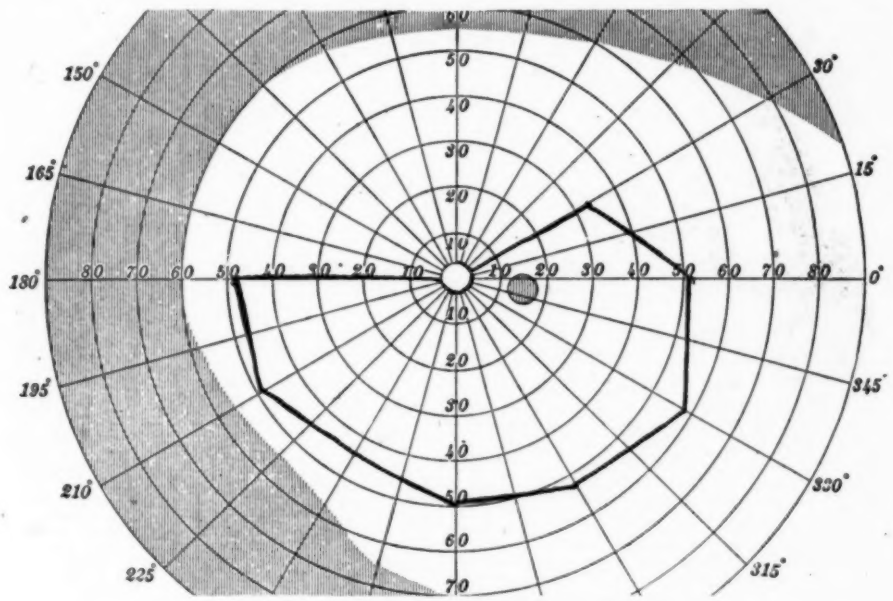

Fig. 2.—Visual field Oct. 4, 1924, Morton perimeter.

The material available for pathologic examination consists of the outer (or temporal) portion of the globe essentially circular in outline on its cut surface and measuring about 23 mm. in diameter. The nervehead is not on the segment and the anterior portion is cut below the level of the pupil. The thickness of the hemisphere is about 11 mm. The outer half of the hemisphere was embedded in celloidin and from it sections were cut and stained with hematoxylin and eosin.

In the hemisphere which was not

used for embedding, there is detachment of the inner coats of the eye except at the ciliary margin and at the pole opposite the cornea, at which place retina, choroid and sclera are all united. Whether the loosening of the coats occurred in vivo or was entirely or in part the result of postoperative manipulation, fixation, and cutting cannot be determined. The impression is that it occurred in large part ex vivo. At the pole opposite the cornea is a light grayish lenticular shaped mass in apposition with the sclera and the retina. A distinct choroid element between them is practically indiscernible. This lenticular mass has a length of about 10 mm. and a thickness of 2 mm. at its center. It gives the impression of being serous fluid coagulated by the fixing fluid (solution of formaldehyd). Between the thinned caudad edge of this lenticular mass and the sclera, is seen a thin (0.5 mm.) reddish brown streak extending caudad for about 5 mm. from nearly the pole opposite the cornea. This narrow reddish brown streak seems to represent the outer edge of the hemangioma of the choroid, the bulk of which was in the nasal half of the globe which was used for sectioning.

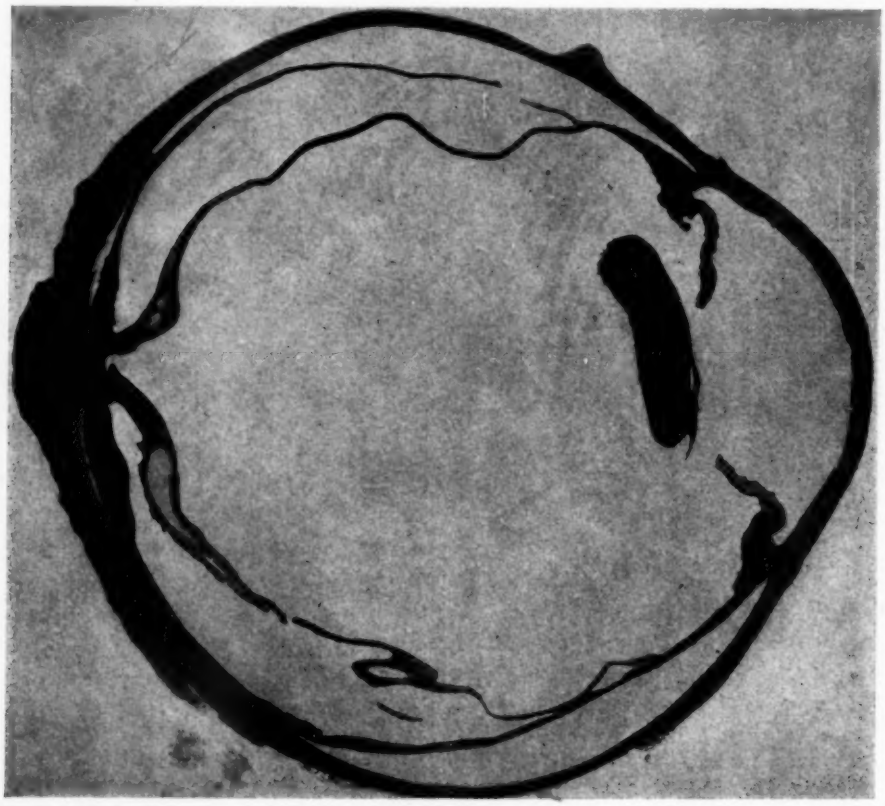

Fig. 3.—Saggital section of eye. Tumor seen on either side of nervehead, slightly separated from sclera. The retina is detached thruout, much separated from the tumor; and, in the portions overlying the tumor, shows much thickness and vacuolization.

The appearance of the eye in vertical section is shown in figure 3, in which most of the characteristic lesions may be seen. Anterior to the ciliary region the section appears normal. The retina is seen detached from the entrance of the optic nerve to the ciliary body. The tumor is seen lying between the detached retina and the sclera, from which it is slightly loosened, especially at the base. The hemangiomatous mass extends caudad

of the nervehead about 5 mm. and cephal about 3 mm. The maximum thickness of the tumor is imperceptibly in excess of 1 mm. occurring in the portion caudad of the nervehead. The tumor appears to take origin from either side of the nervehead. It is of comparatively simple structure, being a hemangioma of cavernous type. (Fig. 4.) The blood sinuses are not round for the most part but appear to be compressed in direction parallel with the coats of the eye. The smaller sinuses

Fig. 4. Middle of caudad portion of tumor showing simple cavernous structure of growth. About 150 diameters.

are more nearly round and have a diameter of about 150 microns, while the largest ones measure from nearly 600 in length to 30 and 60 microns in breadth. None of the sinuses contain thrombi. Many of them are empty, the impression being that they are devoid of red cells because these have dropped out in preparation of the slide, rather than that the comparatively few empty sinuses are lymph channels. Very little connective tissue is present in the tumor. Usually the endothelium of each sinus is in direct, or almost direct apposition with the adjacent sinus. No bone formation is found anywhere in the sections. Comparatively few pigment cells are present. Almost none occur in the interior of the tumor, they being found along the inner and outer borders of the tumor, as tho they had been originally parts of the normal choroid. In the cephalad segment of the tumor, however, and around a rather large blood filled sinus, a considerable number of pigment cells are present.

Caudad of the entrance of the nerve thru the sclera there are some dilated vessels and apparent hemorrhage into the episcleral tissue well shown as dark spots in figure 3. There is nothing to suggest that this collection of blood is angiomatous in nature.

As may be seen in figure 3, the peripheral portion of the choroid has separated from the sclera.

Marked changes are noticed in the retina in the portion immediately overlying the hemangioma. Vacuolization and swelling are so great that they are readily seen in these portions of the retina in figure 3. The greatest thickness of the retina in the vacuolated place is 900 microns, while the thickness of the retina peripherally and where it appears normal is 150 microns. In the cephalad swelling of the retina the vacuoles are mostly rounded, the largest measuring 150 microns and the smallest about 15. (Fig. 5.) In the caudad swelling, in addition to the one large elongated vacuole having a maximum width of 65 microns and a length of several millimeters, there are rounded vacuoles ranging in size from about 20 to 130 microns in diameter.

At the extreme periphery of the retina cephalad and caudad just before its attachment to the ciliary body are a few small vacuoles varying in size from 25 to 50 microns. The vacuolization takes place between the outer and inner molecular layers. Usually where it is most marked and where the retina is most thickened, the normal arrangement of these two layers is much interfered with, the nuclei reduced in number and scattered and the layer of the rods and cones not distinguishable as such. (Fig. 5.)

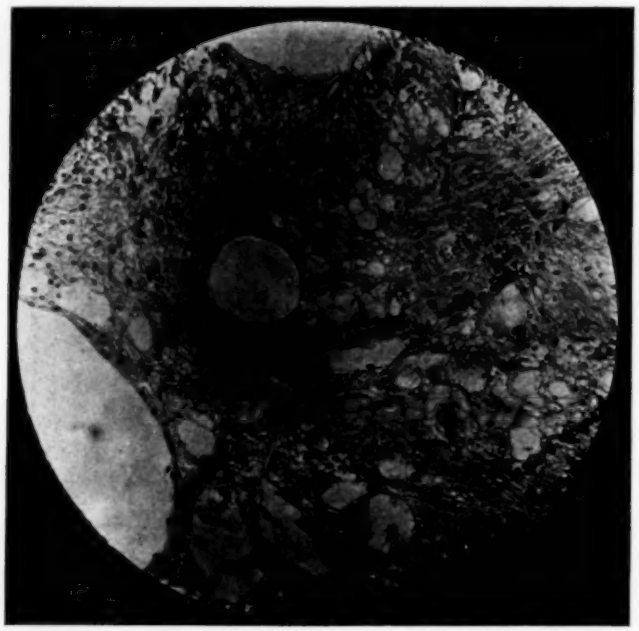

Fig. 5. Swollen and vacuolated portion of retina. The large nuclei at lower edge of field represent the ganglion cell layer. The fairly well defined nuclei just beyond represent the inner molecular layer. The outer molecular layer is represented by the scattered nuclei in the upper segment. The small concave edge in the upper portion of the figure is part of the free edge of the retina. The larger concave edge at the lower left hand border is the edge of a large vacuole. About 100 diameters.

These changes in the retina are not accompanied by inflammatory cells, nor are inflammatory cells found in the hemangioma itself. The conspicuous fold enclosing an apparent vacuole in the caudad portion of the retina, midway between the ciliary body and the nerve entrance (Fig. 3), is apparently an artefact due to looseness and folding of the retina, as that apparent vacuole is between the nerve fiber and ganglionic layers and is not between the molecular layers, as is the case of the other vacuoles.

SUMMARY.

What is believed to be the 26th case of hemangioma of the choroid, as demonstrated by biopsy, is reported.

An analysis of this and 25 cases previously reported in the literature is given including the history of the disease, its etiology, pathology, symptomatology, course, prognosis and treatment.

Hemangioma of the choroid appears to be a rare intraocular tumor, but probably systematic microscopic examination of all enucleated eyes would show it to be more frequent than reports indicate. It is most frequently observed during the second decade, but may occur at any age. It does not appear to grow rapidly but ultimately causes detachment of the retina and glaucoma. The tumors observed have not been invasive in character nor caused metastases. Diagnosis from other intraocular tumors is difficult but possible. Treatment consists in enucleation.

122 *N. Lafayette Blvd.*

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PIGMENT CHANGES IN THE CONJUNCTIVA OF THE LIDS IN TRACHOMA.

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Pigment deposits in the conjunctiva are rare. But a number of cases are found reported in the literature and here summarized. Four additional cases are reported, with histologic details observed in two of them. The name "trachoma stains" is suggested for such lesions. Their relation to other local deposits of pigment is discussed From the ophthalmologic service of Prof. Dr. E. Machek in the Government Hospital in Lwów.

The conjunctiva of the dark races generally contains brown pigment. It is darkest in the region of the limbus and the part of the conjunctiva which is most exposed to light in the palpebral fissure. The pigment is in the epithelium, especially in the basal cells (Hauschild, Bourquin, Steiner[1]).

Pigment has been found in the conjunctiva of the light races also, in the conjunctival limbus, (Kazuo, Morax). Redslob has proved by Masson's method, depending on melanin's affinity for reduced silver, and by the so-called "dopa reaction" of Bloch (dioxyphenylalanin), that a substance producing pigment constantly exists in different parts of the conjunctiva. The uncolored cells, producing the pigment, and belonging to the Langerhans cells, are to be found principally in the parts containing the most blood vessels. The epithelium, apart from the blood vessels, does not contain any melanoblasts.

The pigment changes of the conjunctiva as congenital or acquired stains, appear mostly on the eyeball, seldom at the lid margin, often near the fornices, and very rarely in the tarsal conjunctiva. The conjunctiva of the lids, especially the tarsal conjunctiva, does not seem to be a fit tissue to collect the pigment. For instance, in the case of the intensive melanosis of both eyes, published by Kestenbaum, the skin of the lids and conjunctiva of the eyeball were strongly colored, but the palpebral conjunctiva was nearly devoid of pigment.

The dark stains, appearing in the conjunctiva of persons with trachoma, except the pigmented changes described by Steiner and often happening in individuals of dark complexion, appear very rarely in the light races. For example, Wicherkiewicz, during forty years of practice, had only seen 3 cases, described by him in two publications. Before describing the cases seen by me, I shall give a short description of the few cases in the literature, and shall mention ways in which the authors explain these changes.

The pigment stains occurring in the palpebral conjunctiva in cases of trachoma, were described first by Steiner, who observed them in the Malays and Chinese in 1898, and described them in his book in 1906. Such stains were observed by him not only in trachoma, but in other diseases of the conjunctiva, even in normal conjunctiva. The pigment was always located in the epithelium, in cells of an irregular shape and colored edges, often joining into a colored net. The pigment was found in the cellular tissue, only when it penetrated there with the epithelium. As to the small pigment stains in trachoma, he records they often appear in scars on the upper eyelids, and are caused by the action of trachomatous disease on the chromatophores placed, even in normal conditions, in the conjunctiva of these persons.

In 1907, Küsel described pigment stains in two Jewesses, with trachoma, during scarring. In the first case the little black stain, about the size of 4 mm., was in the tarsal conjunctiva of the upper eyelid; in the second case an irregular little stain was over the lower tarsus. They confirmed in both cases, even macroscopically, that the pigment was lying deeply under the epithelium. In the first case, Küsel examined some scrapings, histologically, and remarked, that the brown and yellowish pigment was in a granular shape in the fusiform cells and in the wandering cells with colored edges. These cells were deep enough under the epithelium; but the latter and the cellular tissue close

under it, had no pigment. Küsel did not remark the pigment being especially arranged regarding the blood vessels. The iron reaction was negative. Küsel thinks these little stains come from blood extravasations of the conjunctiva.

In 1911, Victor Reis reported a case untreated, in a woman, who had suffered trachoma with scarring: In the center of the conjunctiva of the upper lid, bordering upon the fornix, there is a dark stain, about the size of 4 mm. The histologic examination showed black granular intracellular pigment, located in the loose fibrous cellular tissue, deeply under the epithelium. These cells were full of pigment, lying close together in black stripes. There was no difference between the colored and the uncolored cells in respect of shape. The author does not confirm the pigment being topographically dependent on the blood vessels. The iron reaction was negative.

For chronologic order, it is necessary to mention here the work of Steiner (1913). Discussing the subject of pigment stains in different races, and the possibility of producing malignant neoplasms, he speaks of the pigment changes of trachoma, the Javanese showing increase of pigment early in trachoma. The pigment is freely gathering on the upper lid, surrounded by the granules of trachoma. When the acute signs have disappeared, the pigment remains in the scar, having the shapes of nets and bows.

The next three cases were published by Wicherkiewicz in 1914. All these cases concern trachoma in the scarring stage. In the first case, a dark irregular stain about the size of 3-4 mm. was seen in the center of the conjunctiva of the upper eyelid. The time of the previous treatment was not mentioned.

In the second case, treated twice before in the clinic, was noticed a dark little stain about the size of a millet seed, in the center of the upper eyelid. Nothing was said about the color of hair and skin, in these cases.

The third case was that of a young Jewess of dark complexion who was never treated. She did not suffer much from her eyes. A little stain, about the size of a millet seed, was seen on the conjunctiva of the upper eyelid. The upper lip had a dark stain, the size of a small pin head. The histologic examination was made with nearly the same results in these three cases. There was only the difference in the grade of the small cell infiltration. The epithelium was without pigment, the masses of black and brown pigment were situated in the deeper layers of thick fibrous tissue containing few nuclei. They formed dark stripes, divided by lines of uncolored cellular tissue. The pigment was lying in the cells as small granules, but chiefly between them.

According to Wicherkiewicz, the little stains of trachoma resemble those on the mucous membrane of the lip, the trachoma causes diffusion and the spreading of the stains. Persons of dark complexion have a great tendency to such stains.

Rosenhauch records a case of pigment staining in trachoma in the white race, briefly mentioning the stain, located in the palpebral conjunctiva of a dark complexioned Jew who had trachoma. But on the other hand, he presented 2 other cases with the stains on the conjunctiva of the upper eyelid without any signs of trachoma. In one of these cases, the stain was large enough, and it looked histologically like a pigment mole. The histologic picture of the second case was identical with that of the first one of Wicherkiewicz. In cases treated by Rosenhauch the latter remarked pigment stain with trachoma changes in one case only, in 2 cases the stain was in the conjunctiva without trachoma. According to the histologic study, Rosenhauch thinks that the pigment stains, observed in cases of trachoma, are identical with congenital stains in different parts of the body, especially on the mucous membrane of the lip not connected with trachoma.

Steiner, in his last work on the pigmentation of the epithelium of conjunctiva and of cornea of the Javanese and Chinese (1923), says when the tra-

choma invaded the limbus, he found, tho seldom, some stains in the cornea. The tarsal conjunctiva had many small stains. The pigment gathering in the acute stage of the disease, becomes mechanically removed with the granules, and the scars arising do not allow the pigment to reenter these places. This agrees with the observation of Redslob, namely that the pigment cells multiply only in the vicinity of blood vessels.

THE WRITER'S CASES.

CASE 1. Moses K., a man of 60 years, suffered from trachoma about 30 years.

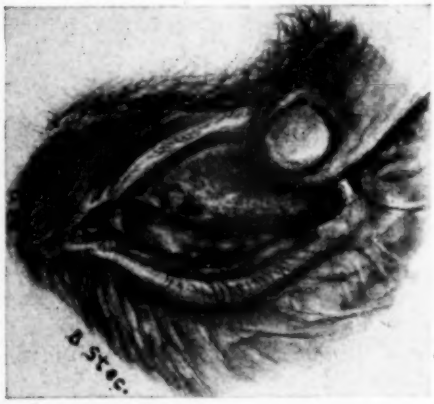

Fig. 1.—Two pigment stains in conjunctiva over posterior edge of tarsus. Case 1.

was treated by private physicians at the beginning of his disease, and then in the section of eye diseases of our Hospital. He also suffered from suppuration of his right lacrimal sac and was treated a short time by probes. Since then, he has not been treated. He never has been seriously affected.

I saw him April 29, 1924, a strong muscular, well nourished man. His hair is mostly gray and black, the skin is dark colored; much pigment scattered on the neck, and in the neighborhood of the armpit and the hip. In other places, there are many gray and bronze stains, close together, often joining. On the face one can see some small, dark, gray moles, and on the trunk there are some little colored warts.

The eyes: The secretion of mucus was scanty, the skin of the eyelids unchanged; the eyelashes regular. Right eye: the cartilage of the upper lid is thickened and curved; the conjunctiva of the upper lid swollen and contains few blood vessels, some irregular scars and many granules of trachoma, easily expressed. There are fewer granules on the conjunctiva of the lower lid and in the lower fornix. The scars are less visible. The eyeball is injected within the conjunctiva and the eyelashes. The limbus is mostly occupied by the scar, in the center of which there is an ulceration. The remaining part of the limbus is covered with an opaque layer, so that the deeper parts are not visible. The whole limbus is a little flattened.

Left eye: the upper tarsus is less changed than that of the right eye. Fig. 1. The conjunctiva contains fewer granules the scars more visible. The largest of them forms a thicker line, along the retrotarsal groove, and two thinner scars come together with the stains described below. The edges of these scars form a thick net implicating the whole surface of the conjunctiva, over the tarsus. In the vicinity of the upper edge of the tarsus, in the limit between the central and nasal part where the scars run together, there are two dark brown stains, close together, of less saturated color in some places, of which the first one, located nearer the inner corner, looks like a small letter "c," the second one looks like a little obliquely located letter "v" and the surface over the stains is smooth and shining. The stains occupy a surface about the size of 6-7 mm. There are some granules in the lower fornix. A film extends out into the limbus from the upper side, reaching half the limbus, and it ends with many small confluences.

The fundus of the eye: In the right, is not visible; in the left is dark. Vision: Right eye, hand motions; Left, counts fingers at 2 meters.

Diagnosis: Trachoma, granular cicatricial, pannus; both eyes leucoma of corneas. Pigmentation of upper tarsal portion of left conjunctiva.

May 16, 1924. Tissue was excised from the conjunctiva with tarsus containing the pigment, for histologic

study. The whole tarsus contains pigment.

Case 2. For this case, I have to thank the kindness of Prof. Dr. E. Machek. Antonia F., 74 years old, married at 19 years, had 16 children, and 8 of them died. When 19 years old, she had smallpox but without ocular complications. She contracted trachoma about 30 years ago. She was treated only one week at the beginning of the disease, and a few days in 1914 and 1918. She bathed her eyes with alcohol and did not use her left eye during 20 years. Three weeks ago, she got influenza, which lasted 2 weeks. Since then she had pain in the right eye, and the sight of that eye is imperfect.

June 7, 1924. A dark complexioned gray woman. The face contains some dark and bronze spots, about the size of a millet grain. Here and there are some bluish hemangiomata. On the lower lip, a little to the left from the central line, is a dark stain, on the mucous membrane in the neighborhood of the skin. The stain is little distinctive, on a dark, bluish ground of the lip.

The eyes: The mucous secretion is a little increased. The edges of the eyelids are slightly swollen and red.

Right eye: The upper tarsus is thickened and bent like a small boat. The conjunctiva of the lids is thin (atrophic), smooth, and a little injected. The subtarsal groove of the upper eyelid, on account of a broad retracted scar, is more visible. In the center of the subtarsal groove, there is a dark, gray, roundish stain, of the same color in the whole space. Fig. 2. The surface of the stain measures 1.5x1.5 mm. The conjunctiva in this place is smooth and thin. We can also see some broad scars, joining one another in that vicinity. We also see a thin net of scars on the conjunctiva of the lower lid.

The bulbar conjunctiva is lightly injected. The cornea is quite uniformly muddy, and contains many stains, running together in the nasal part. A thin film is standing out downwards, reaching half the limbus. The surface of the cornea is not even, and it seems to be stippled all over. Under the microscope we see superficial vesicles, coming from the swollen epithelium. The pupil of the eye is somewhat dilated and does not react to light and convergence. The iris is gray with prominent stroma. The tension of the eyeball is raised: + T. — 1. The details of the fundus cannot be recognized by the ophthalmoscope.

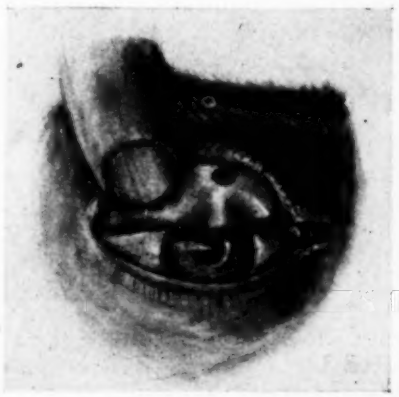

Fig. 2.—Pigment stain of tarsal conjunctiva in Case 2.

Left eye: The tarsus of the upper eyelid is larger and more bent than on the right side. The conjunctiva is scarred and a little more injected, than that of the right side. The subtarsal groove is distinctly marked. Over it, on the temporal side from the center, we notice a flat, prominent spot from which hard yellow masses are projecting backward, the infarcts of the glands. The eyeball is a little injected within the eyelashes. The cornea being injected, seems to be smaller than that of the right side. It is quite muddy and contains many jutting stains, especially in the center. The film is thicker than that of the right side, coming from above, and covering almost the whole cornea. The A. C. is fairly deep, the iris is dark and its structure is not visible. The pupil of the eye is drawn upwards. It is broad and its shape resembles an oblique oval. It shows no reaction to light and convergence. The tension of the eyeball is raised a little. The red fundus reflex cannot be seen even when the ophthalmoscope is used. Vision: Right eye, counting fingers

at 3 m. The left eye has no perception of light.

Diagnosis: Cicatricial trachoma, pannus, maculae corneae. Glaucoma chronic O. U. Pigmentation of tarsal conjunctiva of right upper lid, super O. D.

The glaucoma was relieved by pilocarpin. Iridectomy of both eyes was successfully done, June 18th. The increased tension of the left eye remained until the patient left the hospital. The pigment stain was excised for microscopic study.

Case 3. Anne W., 58 years old, had smallpox in her childhood. About 19 years ago, she had pneumonia. Later her kidneys and urinary bladder became affected. 12 years ago, she had trachoma. At the beginning she was treated by a physician 8 days, then she used the prescribed drops and ointment. During her illness she was treated by a physician 3 or 4 times. The sight of her left eye grew worse in 6 months and the right eye grew worse a month ago.

June 17, 1924. A dark complexioned woman. Her face contains signs of smallpox. The skin of her face, trunk, hands and legs contains many small bronze stains. Besides them one can see here and there some colored hairy moles. Some of them are elevated. On the breast are some small hemangiomata. The exterior of the eyelids is not changed. The eyelashes of upper lids at the external canthus are turned towards the eyeball.

The right eye: The conjunctiva is thick, smooth and injected a little. The tarsus of the upper eyelid is bent. In the subtarsal groove, a yellowish scar deeply drawn in, is visible. The conjunctiva of the lower lid contains a thick net of superficial scarrings. Some of them running transversely are visible in the vicinity of the fornix. In the lower fornix, near the eyeball, in the limit of the central and external part, a little dark gray stain is located, looking like a letter "y" laid down. Its lower side is thicker than the upper one. The thinner side, containing the letter's handle, is 2 mm. long and the thicker one comes to 1 mm. The colored surface measures 1-1.5 mm.

The cornea is quite cloudy, and contains many stains, and superficial blood vessels. The iris and the deeper parts are unchanged.

The left eye: The tarsus of the upper eyelid, and the conjunctiva, are changed in a similar, but stronger grade than that of the right eye. The scar over the subtarsal groove is larger and more retracted.

The eyeball is not irritated. The cornea is covered with thin haze. In the nasal part, we see a large scar grown together with the iris, thin in the middle and swollen a little.

Vision: The right eye 5/60; the left eye hand motions.

Diagnosis: Trachoma, cicatricial, pannus and maculae corneae. Pigmentation of the lower fornix, and conjunctiva of the right eye. Adherent leucoma in the left eye.

June 23, the operation for trichiasis by the Spencer-Watson method was performed on both upper eyelids.

Case 4. H. D., a Jew of 41 years, who had suffered from trachoma about 6 years. At the beginning of his disease he was treated by a physician during a fortnight. Since then he was not treated and did not suffer. About two years ago, his eyes became worse, so he was treated 4 months by a physician.

A dark complexioned man. There are some small spots on the skin of the face and trunk. Besides many other signs of trachoma, we notice on the right eye the following: On turning back the upper eyelid, a net of scars appear which are conspicuous on the blue and red ground of the conjunctiva. The scar in the vicinity of the subtarsal furrow is most visible. Near the upper edge of the tarsus, in the limit between the nasal and the middle third, we find in the scar a small, round, dark gray stain of 1 mm. diameter. The limiting of this stain is not sharp and the surface over it is smooth.

Diagnosis: Cicatricial trachoma, pannus and ulcer in both eyes. Pigmentation of conjunctiva of upper lids.

HISTOLOGIC CHANGES.

The histologic inquiry was made in the first 2 cases, with following results:

Case 1. The shred is covered with a flat epithelium, of many layers, thicker in some places. The cells of the epithelium have no pigment. Under the epithelium, in the adjoining cellular tissue, there are some fresh typical trachoma granules, without pigment. Here and there, the cicatricial cellular tissue shows some thick infiltration of lymphoid cells.

Case 2. There is a flat epithelium of many layers, thickening and deepening in some places, the trachoma deepenings, in which we find globules of fat, colored red by Sudan. We also notice small fat globules here and there, in

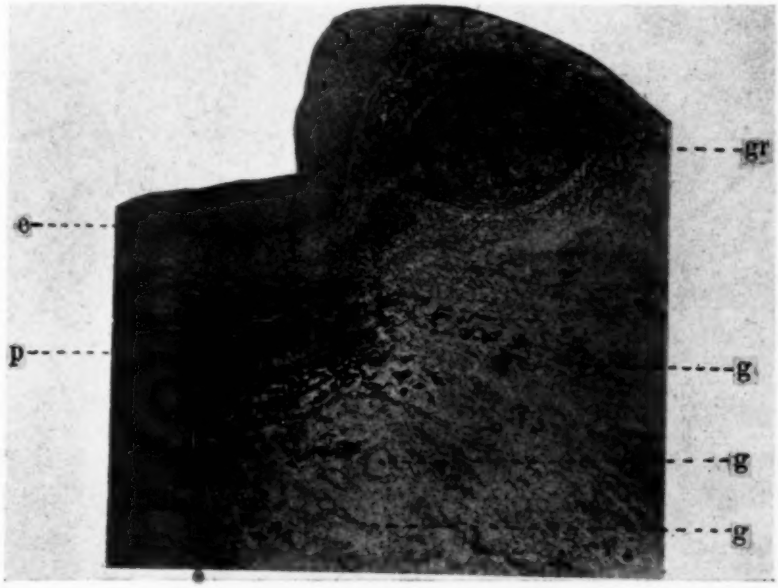

Fig. 3.—Section thru conjunctiva and tarsus of Case 1. e, epithelium; p, pigment deposits; gr, granule; g, blood vessel. (Zeiss obj.B.oc.1)

The deeper parts of the neighboring cellular tissue contain numerous clumps of pigment, on a surface that is not distinctly limited, the pigment being scattered, coming up to the surface almost to the epithelium, while the mass of pigment is lying in the mucous membrane proper and in the tarsus. In the pigment focus, and in the vicinity of it, many widened blood vessels are seen, full of blood cells. The pigment forms dark brown stripes, running parallel with the epithelium, and chiefly arranged along and around the small blood vessels. This pigment is lying in the cells. It fills the cells of the adjoining cellular tissue and epithelium.

In some places the pigment cells form larger spots. In spots containing less pigment, especially in the border, it consists of small yellowish and brownish colored granules, forming larger masses. In a few places there are globules of fat, colored red by Sudan. The iron coloring gave a negative result.

the pigment center, outside the cells. There is no epithelium in some places. Under the epithelium, the adjoining cellular tissue is very thick, sometimes cicatricial and containing few blood vessels and nuclei. In other places, we see many blood vessels with epithelium. On a limited space, we notice the focus contains brown pigment. This granular pigment fills either the cells of the adjoining cellular tissue or the endothelium of the bloodvessels. We find it also outside the cells. The interstitial cellular tissue is surrounded immediately by the blood vessels. Some of the cells are so full of pigment, that they look like dark stains, often making larger blotches. In the periphery, we see little pigment, in some

epithelium and cells of the adjoining cellular tissue. The iron test gives a negative result.

Comparing the pigment changes, described by Steiner, with other cases, we notice their clinical and histologic difference. The changes described by Steiner must be separated and a special group made of them. We notice these pigment changes in the colored races, whose conjunctiva is brownish, even in normal conditions. Generally it contains some smaller or larger stains. The cardinal character of these stains is the pigment localization in the cells of the epithelium. This ectodermal pigment is multiplied in trachoma, as in other inflammatory conditions. The topographic arrangement of pigment clumps is changed by the disease process of trachoma, or by its treatment.

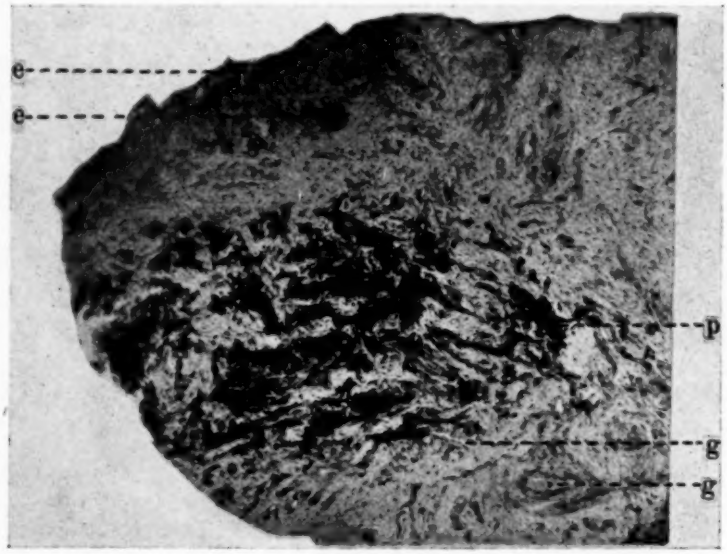

Fig. 4.—Section thru conjunctiva and tarsus of Case 4. e, epithelium; p, pigment; g, blood vessels; (Zeiss obj. B.oc. 1)

COMMENT.

The pigment changes of trachoma, occurring in the white race, and described by Küsel, Reis, Wicherkiewicz, Rosenhauch and in the present 4 cases, (11 cases altogether) form a special group. They have some common and regularly appearing signs, requiring a special name, tho their causal connection with trachoma cannot yet be quite explained. These changes may be be called "the trachoma stains."

We can describe "the trachoma stains," as small, limited, pigment stains, colored brown or black. We can see them in trachoma in the scarring on the palpebral conjunctiva. The adjoining cellular tissue being scarred, contains some pigment clumps under the epithelium, or in the tarsus; but the epithelium has no pigment. The latter consists of some small granules of brownish or dark brown color, and it is lying intracellularly in the cells of the cellular tissue, and of the endothelium of the blood vessels. When the pigment appears in a large mass, it is located extracellularly also. These stains are to be noticed in persons of dark complexion, with tendency to pigmentation.

What is the origin of this pigment? And what connection has it with the trachoma process? There are different answers to these questions.

Küsel says the trachoma does not immediately cause the development of the pigment stains. It only makes bad conditions of resorption by scarring; and consequently the escape of blood into the conjunctiva, which happens during the squeezing of the granule.

This becomes disorganized and turns into the brown pigment. The constant negative iron test in 7 cases opposes such an explanation. Besides, as Reis remarks, the trachoma stains would appear very often after the mechanical removing of the trachoma granules. In 2 cases, one of Reis, and three cases of Wicherkiewicz, the patients were not treated and there was no reason for blood extravasations.

trachoma, and they formed these stains, visible only when the disease had run its course. He says, like Steiner, that these stains arise by increase of the existing pigment. The pigment changes of Steiner's cases appear in the earlier stages of trachoma, around the granules, while the trachoma stains of the white race are visible only in the third stage. Wickerkiewicz found in one case, in a dark complexioned

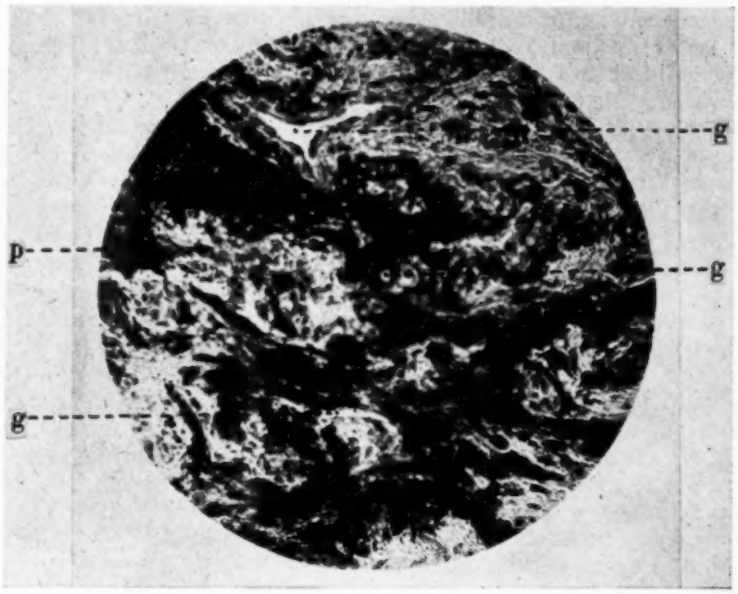

Fig. 5.—Part of same section as Fig. 4. more highly magnified. p, pigment; g, blood vessel; (Zeis, apochromat. 4 mm. project. oc. 4).

The pigment lying intracellularly in all the preparations, also opposes the theory which account for the pigment as proceeding from the blood extravasations. It shows the pigment first accumulated in the cells and then, during destruction of the cells, it was drawn into the interstitial tissue.

Wickerkiewicz, like Küsel, regards the trachoma as not being the immediate reason for production of the pigment stains. He supposes, that the pigment was in the conjunctiva independently of trachoma, in very small stains, difficult to notice. He sometimes noticed it in different parts of the conjunctiva without any dependence on the trachoma, or he saw single chromatophores that became multiplied by the inflammatory process of

woman, a colored patch on the lip, noticed by the patient some months before. Therefore, he thinks that a similar "nevus" could arise on the conjunctiva also. The cellular tissue disappearing during the regression of the trachoma caused it to be more visible. The stains of the lip were not examined histologically; but we know that the pigment of the stains is generally located in the epithelium, or in the cuticle; and thence it penetrates into the cellular tissues. In the 7 cases, they did not find any pigment in the epithelial cells.

Besides my second patient, who would not allow the stain of mucous membrane of the lip to be cut out, I have noticed a similar stain on the mucous membrane of the lower lip in

a patient with the changes in the third stage of trachoma. But I did not notice any pigment changes in the conjunctiva. The shred, which was cut out, has been examined in the anatomic-pathologic institution of our University by Dr. Schusterowna, with the following results:

Register No. 604. Isaak L., 58 years old. The shred of the mucous membrane, excised from the lip is covered with much thickened, flat epithelium; and one can find much pigment in its basal cells. Many chromatophores are visible in the cellular tissue under the epithelium. This result of histologic inquiry does not speak in behalf of the affirmation of Wicherkiewicz and Rosenhauch, who think that the stains of trachoma are identical with the congenital stains. The pigment stains of trachoma have no connection with these changes.

Pigment stains in the mucous membrane of the lip are rather frequently seen with indivdiuals of dark complexion, especially in elderly people; while trachoma stains appear only rarely. The coincidence of both changes only makes us infer the greater richness of pigment and a tendency to create pigment stains in the mucous membrane.

I think, the two cases of Rosenhauch without trachoma do not prove these stains are not in any causal connection with trachoma. The numerous patients treated by Rosenhauch do not prove it. Among that number of patients, he noticed in only 3 cases the pigment stain on the conjunctiva of the eyelid; and among these, he found only 1 case of trachoma. We must not be surprised, that among such a great number of patients, only two cases happened to represent the very same conditions, nevi. The stains in trachoma, may also be congenital.

As to the relation of the trachoma stains to the coloring of hair and complexion of given persons, we notice. that the hypothesis of Wickerkiewicz about their appearing especially in the dark complexioned men, was correct, altho he reports it in only one case and it was not noticed in two other cases. The patients in my cases and in the case of Rosenhauch, were dark complexioned men with a distinct tendency to stains on the skin.

The mode of explaining the subject by Reis is most probable. Relying upon the inquires of Nencki, he supposes that the trachoma granules in uncured cases become slowly disorganized, and that the melanin proceeds from the albumin of dying cells. Really, 2 of the 11 cases were not cured; 1 case of Reis and 1 of Wickerkiewicz. No data were given in 3 cases, 2 of Küsel and 1 of Rosenhauch. The other cases were not cured in the course of many years.

But that theory does not satisfy us, because it does not explain why these changes come so rarely. The trachoma is often healed without being treated and scars remain. If we wish to explain the pigment stains in cases of trachoma, we must answer the following questions: 1. What is the origin of this pigment and to what kind of pigment is it to be counted? 2. In what relation is it with trachoma? 3. Why does it appear only in the cicatricial stage? 4. Why do we mostly find it on the upper tarsus? 5. In what way is the rarity of these changes to be explained?

The first point is difficult, as the subject of the pathologic and normal pigments, their names and kinds, the way and place of their formation and the chemical composition, are not yet sufficiently explained. The opinions of different authors are dissonant and even contradictory.

According to the argument of Brahn and Schmidtmann, the endogenic pigments are divided into 2 groups:

a. Those coming from the blood pigment.

b. Those arising from some other tissues, autochthonous, or melanin. The last ones are divided into the proper, or epithelial melanin; and into the lipomelanin, which was formerly called lipofuscin, the product of retrogressive degenerative changes. These two forms of pigment, distinctly separated before, have shown themselves chemically identical; and the difference between them is in the manner of oc-

currence and of different lipoids contained in them. Brahn, Schmidtmann, Salkowski, Kutschera, Aichbergen. The melanins in contrast to the hematogenic pigments, contains no iron. As stated above, our pigment probably does not come from blood extravasation. After excluding the epithelial melanin, we must think of them as the lipomelanins.

There are different opinions as to the origin of the brown pigment. Some authors think it comes from ectodermal cells exclusively; others say from the mesodermal ones. Meierowski and Schmidt agree upon the two modes. But they think, there is a cardinal different between them, not only in respect to the place but in the mechanism of their production. While the first pigment is produced in the nuclei of the pigment cells, the second one arises by the influence of light from the pigment of blood, either in the cellular tissue, in the smallest blood vessels, or in the fusiform cells infiltrated by the blood pigment.

Redslob remarked that the melanoblasts are in close connection with blood vessels. They are only in the vicinity of the blood vessels. By their location, they seem to mediate in the exchange of material between the blood vessels and the cellular tissue, where the blood vessels do not reach. One can see a certain connection between the pigment and blood vessels of my preparations. The blood vessels of the pigment center and its surroundings are increased. The pigment is arranged along the blood vessels and in their endothelium.

As to the chemical origin of the melanin of the skin, Kreibich thinks it is produced from lipoid of the cellular nuclei, which can be recognized in this pigment by Sudan III. Hueck, Bloch and Meierowski are not of the same opinion. Kreibich thinks the melanin does not proceed from the fat, but only the lipofuscin is of such origin. But the melanin proceeds from the albumin. During the breaking up of the cellular albumins and production of the melanin, the lipoid is separated as a part which is connected with the albumin in a living cell. During the production of pigment, provided the process is not complete, we get a distinct fat reaction of red coloring, when Sudan III was used. In our preparations, we notice some small groups of oil drops, which show that the process of pigment production is not yet quite finished. The pigment cannot be considered a real melanin or lipomelanin, because they both contain fat.

Bloch thinks the skin's brown pigment comes from the circulation of the substance producing the pigment in the fluids of the cellular tissue, and it turns into brown pigment by the oxygenic ferment (dopa-oxydaze) which is contained in the melanoblasts. That ferment being present only at the embryonal production of pigment, except in the chromatophores of the choroid, appears sometimes, for instance, during the development of colored new growths, melanoma.

Considering the theories spoken of, the pigment of the trachoma stains is noticed as melanin or lipomelanin, arising in the cells of the cellular tissue, in the vicinity of blood vessels, from blood pigment; but not as a result of disorganization of blood extravasations, or from the chromogenic substance circulating in blood. In place of light as an activator, we must assume the action of some oxydase according to Bloch's theory.

There is some analogy to the trachoma stains in the melanosis of the intestines, that appears in old men after inflammatory conditions. Similar pigment masses of the mucous membrane are formed here too. The brown shapeless pigment, giving no iron reaction, is gathered here in the cells of the cellular tissue, or in the wandering cells. The epithelium and the lymphatic vesicles have no pigment. Hueck, one of the best judges of the pigment subject, thinks that these masses of pigment come from the uncolored propigment. The latter is produced by blood and lymph, and it penetrates, by excretion, into the mucous membrane, where it joins with oxygen and turns into brown pigment.

As to the connection with trachoma, only the scars and probably the blood vessels, seem to have an influence on the production of stains. The mechanism of this production is as follows: If a person, whose cellular tissue, blood, or lymph, contains the increased, chromogenic substance, tho within physiologic limits, becomes more affected, during the scar formation, his blood vessels are widening in the scars and even new ones are produced, on account of the inflammatory state. Then the blood circulates more slowly for a long time, in that limited space, which helps an oxydasis, produced by the disease, to give rise to the pigment. This could explain the production of stains, in a small, limited space, but not in scattered form. If we assume the necessity of scarring, during the producing of stains, we have the answer to the fourth question; because the thickest cellular tissue is located in the tarsus and especially in the upper tarsus, and the largest scars are produced there.

The rarity of the appearance of stains shows, that some special influences which operate at the same time are necessary for the production of stains. A general increased content of pigment, or propigment, in the organism tho within physiologic limits; and the tendency to make pigment stains, seem first to belong to these influences. The second condition is fulfilled by scars, considerably diminishing the local exchange of material, making more difficult the resorption and facilitating the deposit of secretions. The acute and increasing process of disease at this time and the local multiplying and widening of blood vessels, form the second condition. These conditions appear, if the patient is not cured for a long time. We also assume the action of some ferment.

Last, we must mention, tho it appears strange, that in spite of the rarity of these lesions, each author describes 2 or 3 cases. We became accustomed in the clinic to reappearing cases, but this multiplicity suggests the suspicion, that these small stains often happen, altho we do not notice them. All efforts to explain pigment stains, in trachoma, are only suppositions and conjectures until the general subject of pigments shall be sufficiently explained. Perhaps the progress of the general science of pigments will explain these changes.

I return many thanks to Dr. Schusterowna of the University, for her kind account of the histologic preparations and many thanks are due to Dr. W. Reis, for his microphotographs.

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PARESIS OF CONVERGENCE FOLLOWING ENCEPHALITIS LETHARGICA.

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SEATTLE, WASHINGTON.

Attention is called to paresis or suppression of convergence as a late sequel of epidemic encephalitis. Seven cases are reported in which the weakness of convergence was discovered months or years after the encephalitis, and was not accompanied by weakness of any particular ocular muscle. Read before the Pacific Coast Oto-Ophthalmological Society, April, 1926.

Every new disease or pathology that comes to our ken, brings with it its retinue of complications, and leaves behind it its debris of sequelae. Sometimes the connection between the original pathology of the disease is quite evident, at other times quite obscure, and occasionally, where the connection might be quite definite, it is overlooked because the sequela is out of the field of the man who treated the original condition, and it does not come to his notice at all.

During the epidemic of encephalitis several years ago, every angle of the eye complications were observed, studied and written into the literature. The original squints, the optic neuritis and other immediate complications or accompaniments were all noted.

However, some of the later eye sequelae, coming on as they do a considerable time after the acute encephalitis is past, have been overlooked, and especially not recognized as a sequela of encephalitis. It is to one of these that I wish to attract your attention. Namely: A paresis, or better, a suppression of convergence. I say suppression because I can hardly call it a paresis, in as much as there is no paresis, but rather a suppression of a physiologic or psychologic function.

The first case came to my attention a little over three years ago, and after working out this case, I appreciated that many others have escaped me. I am citing the following cases, because they have a clear history of encephalitis and are typical examples of this condition.

CASE 1. Mr. H. V. W. About 40 years of age. Up to time of his encephalitis was an active and very successful insurance salesman. Two years ago he had a clear cut case of encephalitis, so diagnosed by his family physician, diagnosis corroborated by one of our best internists.

S. P. At the present time he has a slowness of speech, Parkinsonian facies and comes complaining of occasional diplopia.

R.v.=6/10, $+1.00 \bigcirc + .75$ ax 90°=6/6.
L.v.=6/10, $+ .75 \bigcirc +1.00$ ax 90°=6/8.

At this time he only squinted occasionally.

P. P. C. Remote. Exophoria from 8 to 12 degrees at 6 m. and exophoria of about 18 degrees at 30 cm.

Tropometer readings did not show a clear cut paresis of any muscle. In the hope of relieving this troublesome diplopia, one of my confreres did an advancement of the left internal rectus, and at the present time the squint is more constant, altho the diplopia is a little less troublesome as the patient has learned to suppress the false images.

I feel that this man has a squint amounting to the angle of initial convergence.

CASE 2. Mrs. R. L. McN. Age 31. History of encephalitis two years previous. As she began to recover she had a slight ptosis for two or three weeks but did not notice diplopia. About a year ago she had a cross eye sensation and inability to judge distance.

S. P. Anterior segment, normal. Pupils equal and react to light and accommodation.

R.v.=6/5. L.v.=6/5. Movement of eyes does not appear restricted.

P. P. C. Remote. Exophoria in distance of 20 degrees and a diplopia at near point. Tropometer readings are apparently normal.

This is another case where not only the convergence of accommodation is gone but the initial convergence of paralellism is also partly lost, with consequent potential divergent squint.

CASE 3. Mr. Leslie H. Age 39. Railroad brakeman. Had an attack of encephalitis two years previous. Comes complaining of an inability to read.

S. P. Face shows some loss of expression but is not definitely Parkinsonian.

R.v. 6/8+.25=6/6. L.v. 6/8—.50 ax 45°=6/6. P. P. C. 150 mm.+. Exophoria 14 degrees at 30 cm.

Tropometer readings are normal in every direction, and I made a diagnosis of loss of convergence due to encephalitis.

Two years later this man's condition was about the same.

CASE 4. Mr. R. K. Age 48. First seen in 1923 complaining of difficulty in reading because of diplopia at a near point. Had an attack of encephalitis in January. Diplopia was initial symtom.

S. P. External examination, negative. Anterior segment, normal.

R.v. 6/25—1.00⌒+2.00 ax 30°=6/5.
L.v. 6/16+0.75=6/5.

P. P. C. 350. Exophoria 12 degrees at 30 cm.

Later: September 14, 1925.

Muscles: P. P. C. 400. Exophoria 5 degrees at 6 m. and exophoria 22 degrees at 30 cm.

Tropometer readings showed normal muscle power in every direction.

This patient has binocular vision for distance but can not converge to near point, and has diplopia at reading distance.

CASE 5. Mrs. E. W. Age 53. Housewife. Patient came to me first in December, 1924. Had encephalitis lethargica two years before. Comes complaining of inability to read or sew in comfort.

S. P. Face is absolutely devoid of expression; the speech and mental processes are slow. Anterior segment, normal.

R.v.6/20—0.75⌒+1.25 ax 90°=6/5.
L.v.6/16—0.75⌒+1.25 ax 90°=6/5.

P. P. C. Remote. Exophoria 4 degrees at 6 m. Exophoria 18 degrees at 30 cm. No diplopia; ocular movements normal in every direction.

CASE 6. Mr. S. N. L. Age 35. Salesman. Had encephalitis in January, 1924. Was in bed for six weeks. Diplopia was the first symptom noticed. Came complaining of inability to read in comfort.

S. P. Parkinsonian face; slow speech and mental processes. Anterior segment, normal; the eye stands out under cover at the near point. Does not get double vision with the red glass but can not bar read.

R.v.=6/6. L.v.=6/6. No diplopia. Ocular movements normal in every direction. Mad. Rod. Esophoria 2 degrees 6 mm. Exophoria 8 degrees 30 cm.

CASE 7. Mr. R .W. C. Age 42. Real estate broker. Comes complaining of discomfort on reading. Facial paralysis two years ago which the neurologist interprets at this time as a mild attack of encephalitis.

S. P. External examination, negative. Face expressionless and speech slow. Anterior segment, normal.

R.v.=6/40—1.50⌒—0.50 ax 90° 6/5.
L.v.=6/40—1.25⌒—1.00 ax 90° 6/5.

P. P. C. Remote near point. Tropometer readings, slow. No paresis of muscles. Marked convergence insufficiency.

I have chosen these cases as I think they include every degree of convergence loss, from the simple weakness of convergence that would interfere with comfortable reading, to a complete loss of even initial convergence of parallelism which would result in a small squint.

Careful tropometer readings have failed to elicit any weakness of any ocular muscle. All the cases have a normal accommodation for their age.

As I have said before, I consider the cases in which there is a divergent squint much the same as if the eyes were in the position of rest. That is, the position of lack of tonic convergence such as we see in sleep or narcosis.

I have no explanation of the pathology. Having talked to some of the leading neurologists and ophthalmologists over the country, I have been unable to get any assistance from them.

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CHANGES IN REFRACTION IN DIABETES MELLITUS.

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From the literature on the subject its leading features are deduced. Four cases are reported which were seen after a sudden marked increase of hyperopia. Among the hypotheses to account for it, that of lenticular change is regarded as most probable. Read before the Pacific Coast Oto-Ophthalmological Society, April, 1926.

Eye complications in diabetes are frequent, but among the rarer and more interesting phenomena, are the changes in the refraction occuring during the course of the disease. In this report I refer particularly to hyperopia. Myopia is fairly frequent: (Van Noorden, quoted by Joslin, gives its occurrence as 21%). Hyperopia, on the other hand, has been much less frequent. From a very comprehensive review of the subject published by Roberts in the American Journal of Ophthalmology in 1923, and from the more recent publication by Duke-Elder in the British Journal of Ophthalmology on the same subject, there have evidently been reported about fifty cases of this condition.

Due to the production of rapid changes in the metabolic processes in diabetes, since the application of the results of the recent intensive study of this disease which culminated in the discovery of insulin, I feel that the occurrence of transitory hyperopia in diabetes must be much more common than formerly.

A brief review of the literature shows that the condition has been discussed, at infrequent intervals, by writers of note. Fuchs merely states that in some way not clearly understood hyperopia sometimes developes in diabetes. Lundsgaard, in 1911, reported a case in which the patient, a woman, age 50, developed a hyperopia ranging from 2½ to 5 diopters in each eye. The hyperopia developed 15 days following onset of treatment, during which time the urine sugar had been rapidly reduced. The refraction remained the same with or without a cycloplegic. He described what appeared to be vacuoles in the lenses and believed the refractive change to be due to the lenticular changes. The patient recovered. Zentmayer, in 1911, reviewed the various hypotheses which had been advanced to explain the decrease in refraction, and also reported a case in which a hyperopia of 2 diopters developed in a diabetic patient, age 58, in whom the acquired hyperopia was manifestly greater than could be due to a paresis of the accommodation. The increase in the hyperopia coincided with the lessening of the percentage of the sugar in the urine. In this case the hyperopia diminished to 1½ diopters in one year. He also rather favored a lenticular change as a possible cause of the change. Similar cases have been reported by Wölfflin, Wescott and Ellis, and others.

In the four cases reported by Wescott and Ellis, they stated that there was apparently no relationship between the change in the refraction and the amount of the urine sugar. They suggested that some other factor than the amount of sugar in the blood determined the refractive change. Joslin, in his recent book on the treatment of diabetes, refers to the subject of decrease in the refraction and states that not infrequently with the institution of progressive treatment, the vision often temporarily fails. This is due to a change in the water balance in the body and the disappearance of the sugar. These incidentally involve the lens, causing refractive changes. He quotes a case in which the ophthalmologic examination showed a shrinking and flattening of the lenses. A quite remarkable case was reported by Elschnig in 1923. In one eye the lens had been extracted because of cataractous changes, and the hyperopia occurred only in the other eye. Roberts reported four cases, in three of which the hyperopia developed coincident with a decrease in the blood sugar content. He favored a change in the refractive index of the media as a possible cause of the condition. Duke-Elder, in his very complete paper on

the subject referred to previously, also reports two cases in one of which a hyperopia of 9 diopters in one axis, occurred.

In practically all the cases reported, except those of Wescott and Ellis, the onset of the hyperopia always followed a decrease in the sugar content of the blood. One writer incorporates this constant finding into a fixed rule: that the refractive power of the eye tends to vary directly as the sugar content of the blood; that is, there is a tendency to hyperopia with decreased blood sugar and a tendency to myopia with increased blood sugar.

It has been my good fortune to study three cases of diabetes showing hyperopia with a decreased blood sugar, and thru the kindness of two colleagues to report one other similar case.

Case 1. Mrs. J. B. Age 37. On May 26, 1925, the patient was admitted to the Portland Medical Hospital in a condition of severe diabetic acidosis. In the first 24 hours, under dietary treatment only, the blood sugar dropped from .273% to .134%. With this drop in the blood sugar, the patient complained of very blurred distant vision and was unable to read. The patient's eyes were not examined for several days, and during this time the blood sugar, by diet and 40 units of insulin daily, showed a further drop to .116%. On June 5, 1925, the refraction, under a cycloplegic measured:

O.D. +2.25 S.⌒+0.75 Cyl. ax. 80° Vision=6/6.

O.S. +2.25 S.⌒+0.75 Cyl. ax. 95° Vision=6/6.

An addition of +1.50 S. was necessary for reading.

The eyes externally were normal. Tension was normal. The fundi were essentially negative. Accommodation measured two diopters in each eye. Under treatment, the blood sugar was reduced by July 4, 1925, to .084% or normal. At this time, however, the patient was unable to see with the lenses previously prescribed and examination showed:

O.D. +0.75 S.⌒+0.75 Cyl. ax. 80° Vision=5/5.

O.S. +0.75 S.⌒+0.75 Cyl. ax. 95° Vision=5/5.

Reading correction was not necessary. The accommodation was now 3 diopters in each eye. Muscle balance was normal. Convergence 16 m.a. This patient has been under strict control up to the present time and has shown no recurrence of the hyperopia. At an examination a few weeks ago, the vision of each eye was normal without correction. Accommodation 4 diopters. Muscle balance normal. The patient reported, however, that on each recurrence of sugar in the urine she noticed some blurring of vision, which disappeared immediately when she again became sugar free. In this case a paresis of the accommodation could account for the refractive change.

Case 2. Mrs. R. S. Age 56. Was first put under treatment for diabetes on August 19, 1923. Blood sugar at this time was .227%. Between August 19th and August 28th, 1923, the blood sugar was reduced, by diet and 24 units of insulin per day, to .149%. At this time she complained of hazy distance vision, and made the discovery that she could see in the distance better with the reading segment of her bifocal lenses. The patient was wearing:

O.D. +1.75 S. V.=5/60.

O.S. +1.25 S.⌒+0.50 Cyl. ax. 90°. Add. +3.00 for reading V.=5/60. Examination of the globes was essentially negative. Tension normal.

Fundi negative except for mild vessel changes. Manifest refraction showed:

O.D. +4.75 S. Vision =5/4.

O.S. +4.75 S.⌒+0.25 Cyl. ax. 180° Vision =5/4. Add +2.75 S. = J. 1.

From August 28, 1923 to October 31, 1923, the blood sugar was gradually reduced to .086% or normal. During this time the refraction slowly changed, increasing in amount so that two months following the onset of the hyperopia the patient was able to return to her old lenses.

This case represents the typical onset of hyperopia occurring suddenly following a rapid decrease in the blood sugar, and the acquired hyperopia is

manifestly greater than could be due to a paresis of the accommodation.

Case 3. Mr. M. M. Age 57. First put under treatment for diabetes on September 23, 1925. Blood sugar at that time was .226%. Under dietary treatment the blood sugar was reduced to .169% on October 3, 1925. The patient's eyes were first examined on the latter date and he gave a history of good distance vision until a few days previous to this time. Vision at the time of examination was:

O.D. +1.00 S.=5/15.

O.S. +1.00 S.=5/15.

Muscle balance was normal. Accommodation O. D. 1¼ D.: O.S. ¾ D. Manifest refraction was as follows:

O.D. +2.75 S.⊂+.25 C. ax. 175° Vision=5/5.

O.S. +2.75 S.⊂+.25 C. ax. 70° Vision=5/5.

With +2.75 S. added he read J 1.

This patient has remained under observation and treatment for his diabetes but a short time and the ultimate outcome of the refraction is unknown. The case demonstrates, however, the onset of hyperopia following a decrease in blood sugar.

Case 4. Mrs. M. B. Age 23. First treated for her diabetes on December 5, 1925. At this time the blood sugar was .236%. Under dietary treatment the blood sugar in one week's time was reduced to .180%. The patient now complained of being unable to read. Examination by manifest refraction showed

O.D. +0.50 S. Vision =20/15.

O.S. +0.37 C. ax. 180° Vision = 20/15.

The patient required a +1.25 S. before each eye to read J No. 1. There is no record of the accommodation and muscle balance in this case except that the accommodation was normal following the acute attack of the diabetes. One week following the examination of the refraction, the patient was able to discard her glasses and was able to use her eyes as normally as before the onset of her symptoms. At this time she was sugar free. It is manifestly quite probable that this case represents purely a ciliary muscle involvement.

Many theories have been advanced to account for the phenomenon. Some observers hold to the theory of paresis of the accommodation, and this evidently holds true in a certain number of cases. However, in the great majority of cases reported, the greater percentage occurred during the age when the accommodation was not active. Also many cases develop a hyperopia greater than the range of the accommodation would allow.

The theory of shortening of the antero-posterior axis of the eye due to loss of fluid in the vitreous: this does occur in the very advanced cases of diabetes and produces the soft eye of terminal diabetes. However, in this morbid condition the blood sugar is abnormally high, and if the hyperopia occurred in the milder cases, it would be with an increased blood sugar, whereas the hyperopia in reported cases occurs with a decrease in the blood sugar.

The theory of an index change: this possibly could account for some very small changes in the refraction, but it is difficult to see how a change of 8 or 9 diopters, as has been reported, could be produced by a concentration of sugar in the blood, especially as experimentally a solution of sugar must be concentrated by 20% to bring about a change in its refractive power of one diopter. This would mean that enormous amounts of sugar would have to be circulating in the blood stream, far above the highest known sugar value.

The theory of lenticular changes: According to Duke-Elder, there are several considerations pointing to lenticular changes as being the basis for the occurrence of the hyperopia.

1. 81% of cases reported occurred between the ages of 40 and 60 years, the period of lenticular instability.

2. Elschnig found the changes occurring in only one eye, the fellow eye having had the lens removed.

3. Spalding, quoted by Joslin, has noticed a "shrinking and flattening" of the lens.

4. The occurrence of astigmatic changes in 36% of cases suggests deformation of the lens.

5. The sudden yielding of the lens to osmotic forces dependent upon the water balance of the body. We know that the lens is particularly resistant to change, but it is possible that following prolonged assault it may give way to the changes in the water balance of the body; in other words, an osmotic force might be set up and, following the laws of osmotic forces, fluid would flow into and out of the lens, dependent upon the amount of sugar concentrated in the blood. With an increased sugar content, the molecular concentration of the blood and tissue fluids would fall and the lens would be bathed in a fluid of a tension lower than its own molecular state. Consequently there would be a flow of fluid into the lens increasing the refraction and producing myopia. The reverse holds true with a decreased blood sugar. The molecular tension of the blood and tissue fluids is now of a higher tension than the lens and fluid flows out of the lens, decreasing its refractive power thereby producing the hyperopia. The question of the loss of the normal elasticity of the lens, due to the loss of fluid and consequent flattening, even with a normally acting ciliary muscle, is also to be thought of.

Nothing definite can be said as to the real cause of the condition, and undoubtedly many things enter into the question of the disturbance, but the weight of evidence would tend to imply that lenticular changes are paramount in the production of the hyperopia. Refractive changes do not occur in all diabetics, and of those changes that do occur, hyperopia has been comparatively rare. Fortunately, there is always a return to normal and patients can be assured that with continued control of their diabetes, and in the absence of any pathologic retinal changes, the vision will return to its former state.

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TONOMETRY BEFORE CATARACT EXTRACTION.

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Certain complications of cataract extraction appear to be connected with departure of the intraocular tension from normal. Some are connected with subnormal tension and others with hypertension. In the former cases preoperative instillation of atropin may be indicated. In the latter treatment with pilocarpin or a preliminary iridectomy should be resorted to. Read before the Academy of Sciences of Havana, May 14, 1925.

Platner, a disciple of Brisseau, had for the first time called attention to the hardening of the eye when affected by glaucoma. But neither he nor his contemporaries gave any great importance to this sign, that was only interpreted correctly in 1830, when Mackenzie analyzed it thoroly and from such analysis arrived at a logical treatment—sclerotomy. From that date investigation of the ocular tone by digital palpation became a general practice. In 1863, Graefe, Donders and Haemmer separately tried without satisfactory results to measure the intraocular pressure by means of instruments; the same happened to Dor two years later, and to other less fortunate investigators, until 1905, when Schiötz, of Christiania, devised his tonometer.

After that, five or six instruments serving an identical purpose have been invented, and altho any of them serves the end of ascertaining the normal or pathologic tension of the eye, the truth is that at this time we do not know definitely what is the normal tension, expressed in millimeters of mercury, altho all the tonometers claim to fulfill that object. Schiötz indicates as normal all tensions between 15 and 25 mm., whilst MacLean, based on experimental studies, claims that such normality lies between 22 and 40mm.

The investigations I have made with the apparatus of both inventors have convinced me that the limits stated by them are quite admissible with their respective tonometers, which goes to prove that the ratio between their indications as compared with that of a manometer of mercury is not correct.

In order to avoid confusion, I must state that all the quantitative figures given in this report have been secured with the MacLean tonometer; this should be borne in mind by those who use any other apparatus.

The general use of the Schiötz and the MacLean tonometers for the past twenty years has made possible many investigations, especially in connection with glaucoma and iritis, altho many other aspects have also been studied. In our own times, when so much is written in all parts of the world, in every known language, on ophthalmologic subjects, it becomes rather difficult to present any given matter as completely original. But I can at least affirm that I have found nothing, either written or cited, on the theme treated in this report: the advisability of systematically practicing preoperative tonometry of the eyes in cataract extraction.

Whichever may be the procedure for extraction of the cataract favored by the surgeon, viz.: the classic extracapsular, or the intracapsular, after the methods of Barraquér, Smith or Wright, it is certain that with all of them certain accidents frequently occur, during the operation or after it, which affect its outcome, probably in a much larger proportion than that shown by the statistics. In an effort to minimize such disagreeable accidents, I have studied for more than a year the ocular tone of the patients whom I operated upon for cataracts.

All surgeons know that, after having cut into the cornea, different aspects may change the course of the operation. On certain rare occasions the cataract comes out of its own accord, without any further surgical manipulation; in others the anterior chamber narrows appreciably, either before cystotomy or more frequently after it, by the forward displacement of the opaque crystalline, as if it needed only a slight pressure upon the lower part of the cornea to find its way thru the corneal breach. Then again, the vitreous humor shows thru the wound before the time for the extrac-

tion, obstructing it noticeably, and in all these cases the oculist must needs proceed dexterously and cautiously, to avoid losses of vitreous humor. The preoperative tonometric measurements have convinced me that in such cases there exists hypertension or at least one higher than 35mm.

In some instances, on the contrary, the lens remains in place after having cut into the cornea, and is luxated easily when attacked with the cystotome, or else, at the time of the extraction, upon pressing the cornea, the lens, instead of rotating on its horizontal axis and advancing towards the wound, allows itself to be depressed somewhat, forcing the use of risky manipulations. All this is due to the hypotension of the eye, that does not offer the crystalline sufficient support to facilitate its rotation. In my experiences, in almost all such cases the intraocular pressure had been lower than 20mm.

I have found that the most auspicious situation for success is when the tonometer shows between 23 and 33mm.

Another precious indication which we may derive from preoperative tonometry is the adequate use of solutions of atropin. Many surgeons actually employ atropin to facilitate the extraction of the lens; others, the larger number, make use of it immediately after the operation, or after the first few treatments, as a preventive of postoperative iritis, of the adherences of the iris or of capsular fragments, and of hernias of the iris. There are some who decry its use as needless, and even condemn it as responsible for the appearance of glaucoma.

Preoperative tonometry shows the reason for this divergence of opinions. I soon verified the absurdity of the practice, previously followed also by me, of almost systematically using atropin with all patients operated for cataracts, forgetting that we are dealing with individuals whose vascular system is frequently affected, and hence predisposed to glaucoma; and the tonometer has taught me, as stated in the preceding paragraph, that atropin is very useful when the tone is under 28mm.; that when it fluctuates between 28 and 35mm. it may be used very cautiously, and that it must be totally abolished if the pressure goes beyond 35mm. We must not lose sight of these facts during the postoperative course of the cataract.

Moreover, the tonometer advises us wisely regarding iridectomy. The relation of the simple or the combined extraction has been fully discussed long ago, and every occulist has already a set opinion on the subject, either one way or the other. But preoperative tonometry comes to tell us that in all cases where there is hypertension, even in the absence of the diagnosis of glaucoma, we must choose the combined extraction, and that when the tension goes beyond 35mm., previous iridectomy is indicated, postponing the extraction for several weeks.

I never practice suture of the cornea, but those who prefer such practice will find in the hypertension a basis for it.

Conclusions. The intraocular tension of every patient who is to be treated for cataracts must be investigated.

The eye is in best condition for the extraction when the tone lies between 23 and 33m. (MacLean tonometer).

If the tone lies below 23mm., previous application of atropin is useful. In order to operate when it goes above that figure; if it is over 38mm., pilocarpin should be used to lower it to the desirable limit, and if it does not succeed rapidly, iridectomy will be practiced first, followed several weeks later by extraction.

Postoperative use of atropin to prevent iritis, etc., will be indicated when the tone is lower than 28mm.; it may be used cautiously if the tension lies between 28 and 35mm., and it should be totally avoided when it goes beyond the latter figure.

SUDDEN AND TRANSIENT BLINDNESS.

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This paper includes transient monocular blindness associated with hypertension and arteriosclerosis and that recurring in young adults; but not the scintillating scotoma of migrain. Four cases are reported and the common views regarding their etiology are discussed. Read before the Baltimore City Medical Society, Ophthalmological Section, March 25, 1926.

Cases of partial or complete blindness, sudden and transient, are met with which resemble each other in mode of onset—and in their duration—but which are essentially different in their causation and in their course. The conditions to which reference is made do not include the common scintillating scotoma of migrain, nor the visual disturbances due to uremia and other poisons, nor those met with in choked disc, prodromal attacks of glaucoma, etc. The cases referred to in this paper are of two kinds:

I. Transient monocular blindness associated with hypertension and retinal arteriosclerosis.

II. Transient recurring monocular blindness in young adults.

The character of the visual disturbances in these groups, the suddenness of the onset and their unilateral character can only be explained by assuming an interference with the retinal circulation. This assumption is further strengthened by the fact that conditions depending upon stoppage of the circulation in the retinal arteries or their larger branches, namely embolism and thrombosis, are known in some cases to have followed transient disturbances like those described. An interesting clinical and experimental study of the possibility of recovery of retinal function after interruption of the circulation has just been published by Guist.[1] Definite proof has, moreover, been furnished in a few cases by direct ophthalmoscopic observation of marked contraction of the retinal arteries, visible retinal angiospasm.

GROUP I. The following case is typical.

CASE 1. Mr. N, aged about 65, was first seen on account of attacks of dizziness in January, 1921. His vision with correcting glasses was perfect. His eyegrounds showed numerous colloid excrescences of the choroid and very distinct sclerotic changes in the retinal arteries. He was under the treatment of a physician at that time, who informed me that the patient had marked general arteriosclerosis, that the blood pressure was 160/94, and that the urine was free from sugar or albumin. Over a prolonged period he had been taking iodid of potash in moderate doses. On the 9th of April, 1924, he came complaining that about one week before he had obscuration of vision in the right eye lasting a couple of minutes, during which period he was not able to see anything. A few days later he had another attack lasting about five minutes, again affecting the right eye, but this time he thought the left eye also seemed blurred. His vision rapidly cleared up again and he saw perfectly well. This morning he had a third attack in the right eye, lasting the same time. When he came to the office soon afterward his central vision in both eyes was perfect and his fields of vision were normal.

I saw the patient recently (March, 1926) and learned that he has had no further disturbances. The occurrence of several attacks of monocular blindness during a short period, in association with marked general and retinal arteriosclerosis, followed by complete restoration of vision and the absence of further attacks during the last two years, indicates that the attacks were due to angiospasm. The absence of further trouble in this case is remarkable, for most cases which present attacks of this kind in persons with arteriosclerosis end in thrombosis of the central retinal artery or its branches. Wilbrand and Saenger[2] describe these conditions under the heading of prodromal attacks of blindness in arteriosclerosis.

Leber throws doubt upon the view that sudden blindness results from simple primary endarteritis, on the as-

sumption that blindness does not occur until the lumen is so small that no blood can pass thru. He objects on the ground that under such conditions blindness would not come on suddenly as it does, but gradually.

It has been assumed that marked lowering of the blood pressure in arteries whose calibre is already markedly narrowed, may cause a complete collapse of the vessel with loss of function of the retina which it supplies. Such closure may be temporary, the vessel again filling under a higher pressure; or on the other hand, a thrombus may be formed and the blockage be permanent.

The following case is of a different character:

CASE 2. Mr. O, aged 48, consulted me on June 1, 1915. He gave a history of angina pectoris. His physician, Dr. Louis P. Hamburger, has kindly furnished the following note: The patient had been much moved by the impending catastrophe caused by the Baltimore conflagration in 1904 and while standing by, complained of pain in the precordial region radiating down the left arm. From this time he suffered from attacks of angina; when walking up hill or walking fast he was seized with pain in the precordium and interscapular region. The pain compelled him to stand still. The blood pressure varied from 125 to 140.

A few hours before reaching my office, on June 1, 1915, while in his bathroom, he suddenly found that the sight of his right eye became blurred, and was rapidly lost, so that he could not distinguish any object, but was still able to see the light of the window; there appeared to be a bluish haze before the eye. After a few minutes the sight returned, beginning above and soon the entire field was restored. Examination of the fundus showed that the vessels were rather full and showed no irregularities. At the bifurcation of a large branch of a superior temporal artery, there was a glistening white spot as tho a cholesterin crystal were lying on or in the vessel, which showed no other abnormality. Central vision was perfect in both eyes, as was likewise the field.

The patient had no further trouble until November 5, 1917, when he suffered a similar attack. He immediately inhaled nitrit of amyl and the obscuration passed off. But on the following day the attack was repeated. He was examined on November 9 and no fundal lesion whatever could be discovered; the retinal vessels appeared healthy.

The interpretation of this case is not easy. Leber[3] leans strongly to the view that the causes of these obscurations are soft emboli which, he claims, have been observed to pass thru the retinal arteries and disappear. The appearance of the white spot on the bifurcation of the superior temporal artery, referred to above, and which later could not be found, is perhaps suggestive of this explanation.

Angiospasm probably receives more general acceptance as an explanation and Leber himself accepts it for those cases in which the attacks of transient blindness are very frequent. This condition was first brought to the attention of the profession by Raynaud in 1894. Raynaud had himself observed cases with periodic visual disturbances in whom retinal "arterial spasm" was seen.

This subject is discussed in detail in Leber's work. The theory of angiospasm has indeed been taken over from ophthalmology by neurologists. Thus Lewandowsky[4] regards this explanation as applicable to certain cerebral conditions characterized by very transient motor or sensory disturbances.

One of the most remarkable cases in the literature is that of Harbridge[5] with monocular visible spasm of the central retinal artery in a male, aged 49, and frequent attacks of blindness lasting from one to five minutes.

The problem, however, is by no means simple. There are a number of cases on record in which the fundus examined during an attack of transient blindness showed no contraction of the blood vessels, and some in which dilatation was observed. It is perplexing to find no satisfactory explanation.

Leber[6] suggests a spasm of the central retinal artery before its entrance into the eyeball, preventing further inflow into the retinal vessels, but leaving them filled, as is sometimes observed in embolism of the central retinal artery. The consequent lowering of intraocular pressure, he suggests, may result in compression of the vein at its point of exit, which would still further add to the retention of the blood in the retinal vessels.

Rosenstein[7] has lately reported a case in which a man of 28, suffering with mitral lesion, had had frequent attacks of blindness of both eyes with giddiness, "uremia" and vomiting, slow and weak pulse, in which the eyeground showed marked retinal ischemia during the attacks.

Bittorf[8] records pulsating obscuration of vision in a man, aged 65, with marked aortic insufficiency, the patient seeing periodic changes in brightness of external objects, synchronous with his pulse.

Barre and Duverger[9] reported the case of a woman suffering with aortic stenosis and recurrent attacks resembling embolism of the central retinal artery and ischemia of the retina.

GROUP II.

The second class of cases is found in young persons and differs from Group I in the absence of any evidence of vascular disease, either general or retinal.

CASE 3. Mrs. W., aged 34, was first seen September 21, 1922. She complained that for the past 10 or 11 years she had had attacks of flickering which were characteristic of ophthalmic migrain; these attacks had been quite frequent. During the past year she has had 4 or 5 attacks of an entirely different kind; the first in April, 1921, in which the right eye then suddenly became blind for ten or fifteen minutes. During these attacks she had only light perception. In January, 1922, she consulted an oculist, who prescribed eserin, every night and when she had attacks. During the past June she had an attack that lasted fifteen minutes. Between the attacks her sight was quite as good as it had been; there was no pain during the attacks. The examination of her vision showed right eye 20/19 imperfectly; left eye 20/15. She had a marked anisometropia with 3.5 D. of hyperopia in the right and 0.5 D. of hyperopic astigmatism against the rule in the left. The pupils were normal and no other trouble could be found. Her fields of vision on October 12th were quite normal for form and color.

She had no further attacks until April 26, 1923. In the morning there was blurring and dimness; the vision was rapidly lost, but she could still discern a light and the outline of a bright window, but no more. The attacks lasted for eight or ten minutes. The darkness then "broke up" and she saw colors, blue and black. I examined her a few hours later and found the fundi normal, blood vessels normal and tension normal. Nitrit of amyl pearls were ordered. On May 17th she had another attack lasting ten minutes. She immediately used the nitrit of amyl, but it appeared to have no effect. I saw her again a few hours after the attack had entirely passed off and she stated that in these attacks she sees bright lights like an aurora and there are fine lines constantly moving. Her impression is that the sight of the left eye is not in any way impaired during these attacks. Between the last two attacks, there had been a typical migrain attack followed by headache with homonymous hemianopic visual disturbances. On June 13th the next attack occurred. Again for about ten minutes, without complete loss of vision, she could still see the window frame and bright lights. When the darkness "lifts" she sees spots of "King's" blue. Her attention had been directed to the condition of the fellow eye and she was able to state positively that the vision of this eye was not affected.

On July 24th and again on August 27th she had attacks, the former just after a cold bath. The second attack just after dinner; on this occasion she had a headache and aching of her left eye. During this attack she could not see a bright electric light with the right

eye. The attack again lasted ten or twelve minutes or perhaps even longer, otherwise no change. It will be seen that during this period the attacks came on with a fair degree of regularity, once a month. It would be noted that the nitrit of amyl did not appear to give her any relief from the visual trouble, but caused her much discomfort and nausea, and was therefore discontinued. She had no further attacks after September 23rd until May 9, 1924, when a mild attack occurred, during which vision was not lost but objects grew very dim. Recent inquiry (April 15, 1926) has elicited the fact that the patient has had no attack since.

Case 4. Mrs. D., aged 21, was first seen October 21, 1922, complaining of a mist appearing before the right eye on six or seven occasions for about a year at long intervals. The obscuration appears like a veil coming before the right eye, everything gradually gets perfectly black before this eye, the veil then lifts from below. The patient had not timed it, but thought it lasted a minute or two and then the sight is as good as before. The attacks are not followed by headaches, nor has she noted any scintillation. The eyes were examined and nothing abnormal could be discovered; vision in both eyes is perfect. No refractive error. There was no lesion in the eyegrounds; discs and blood vessels appeared to be perfect.

On inquiry I learned from her physician that her health had been quite good and her only complaint was constipation. Her cardiovascular system was normal; her blood pressure was 120/80. Examination of the blood gave a normal cell count. Urine was normal. There was nothing in her general condition bearing upon her visual disturbances. The patient has never had migrain. In January, 1924, I was informed that she had had about three attacks since the past May, varying in intensity. The mother is under the impression that she gets the attacks only when under a strain and when tired and nervous. The attacks have always been limited to the right eye.

On February 13, 1924, she was taken to the hospital for confinement, being 8½ months pregnant. On the day before, she had had an attack of blindness which was not complete and lasted only a few moments. Following her confinement of February 18th, she felt a "quivering sensation," which she believed was ushering in an attack, but the vision was not affected. The eyegrounds were normal. The fields of vision were taken very carefully, for form and color, on April 23rd and found normal.

Her obstetrician gave the following note: "Saw her on November 11, 1923, when she was five months pregnant, at that time her blood pressure was 130/64 and the urine showed no albumin. She was frequently examined; and on February 2nd the urine for the first time showed a trace of albumin, and on February 8th there was considerable swelling of the feet and ankles and the blood pressure had risen to 180/110. At this time the urine had specific gravity of 1010 and 3 grams of albumin to the litre with no sugar. A few granular casts were seen. On February 14th labor was induced because of the definite toxemia. She made a normal convalescence and on April 1st her blood pressure was again 130/70 with but a faint trace of albumin and no casts." Still later, January, 1925, her blood pressure fell to 120/80 and she was in excellent health. The patient informs me (April 10, 1926) that she has not had any further attacks of blindness. She has not had any later pregnancies. The apparent coincidence of the later attacks with pregnancy and their subsequent cessation led to further inquiries; and it was discovered that the patient was married in October, 1921, and that it was then that the first attacks appeared. There is a strong suggestion in this case of relation of the attacks to the sexual function.

The last cases cited are interesting not only because of the frequency of the attacks of blindness, but especially because of the limited period of a few years in each case. In Case 3 there were 11 or 12 attacks during three years. In Case 4 there were 10 or 11 attacks during 2⅓ years and in both cases there has been a long period fol-

lowing their cessation, two years or more.

The occurrence of typical ophthalmic migrain in Case 3 is probably not without importance tho the character of the visual disturbances were entirely different; those of the migrain attacks having affected the sight of both eyes in the typical manner, the other attacks having invariably affected but one eye and that always the same one; in Case 4 also one and the same eye was always affected.

Leber[10] cites a number of cases with migrain, and according to Wilbrand and Saenger[11] spasm of the retinal arteries may occur with migrain. They also cite a number of cases.

The writer was never able to see the patients during their attacks, but any explanation other than angiospasm is difficult to conceive of. We are at a loss, however, to suggest the immediate cause of such localized angiospasm in either of the cases.

In all of the many cases of migrain which have been described to the writer in his practice, the disturbance of vision has been accompanied by scintillation and has been binocular. But there are authentic cases described, in which there was no scintillation and in which the disturbance was monocular. It is quite possible, therefore, that such cases as 3 and 4, in which there was neither scintillation nor hemicrania, and in which the visual trouble was limited to one eye, may nevertheless have belonged to the same group as those of typical ophthalmic migrain. The very differences may find an explanation in the fact that the "paroxysmal nervous disturbance of which headache is the most constant element," as Gowers defines migrain, is characterized in that large group of cases described by Charcot as "migrain ophthalmique fruste," by absence of headache. Is it not possible, that when the paroxysmal disturbance is in the retina, neither hemicrania nor scintillation may appear? This is purely hypothetic. We are obliged, however, to admit the close relationship which the association of transient blindness and migrain suggest. Whytt[12] long ago suggested arterial spasm as an explanation of migrain.

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INCIPIENT GLAUCOMA.

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The early symptoms of glaucoma are often indefinite and rarely characteristic; likely to be neglected or ascribed to eyestrain, or migrain. Thoro examination of the anterior segment of the eye, and the posterior segment with the ophthalmoscope, and careful field taking are necessary to detect the cases which have had no frank inflammatory attack. In the end such cases are recognized and reflect discredit on those who have overlooked the early stages of the disease. Read before the North Dakota Academy of Ophthalmology and Laryngology, May 2, 1926.

In the same ratio that conjunctivitis is confused with iritis, and iritis with glaucoma, among men in general medicine, I believe incipient glaucoma is missed of recognition by the general run of eye, ear, nose and throat men. Careless history taking of refraction cases probably affords the first and greatest pitfall; followed, in their order of frequency, by careless examination of the anterior segment of the eye by daylight and by oblique illumination and loupe in the dark room; next, by careless examination with the ophthalmoscope of the media, nerveheads and retinal vessels; and, lastly, by shirking the task of field taking with the perimeter and campimeter in cases suggestive enough to justify such investigation.

I am sure that most of us underestimate the importance of the symptom "blurring," and the grave possibilities and responsibility of passing lightly over such complaint. To be sure, the blurring complained of in the vast majority of cases may be due to uncorrected astigmatism, convergence insufficiency, or accommodation overload. But lose not sight of the blurring in the periodic occurrence of edematous cornea in incipient or early glaucoma, which may come on almost fleetingly, gives little more inconvenience than blurred vision for a few hours, and recedes unsuspected of the tragic significance it has.

These middle-aged patients often put the matter off indefinitely, with the entirely satisfactory explanation to them-

selves that it is due to a little overuse of their eyes, and that a good night's sleep will straighten them out; as, we know, it generally does. What a pity if these same cases slip thru the hands of the oculist unrecognized, when, as Fuchs says, "early recognition means everything; late recognition means loss of everything." To be sure, this present epoch of flourish on the part of the optometrist, untrained to recognize pathology in the eyes he refracts, is sure to collect a large toll in glaucoma; but, accepting the repeatedly quoted statement of authorities that glaucoma constitutes one per cent of all eye cases coming to us, does not the thought force itself upon us, "Are we not missing and failing to recognize some, at least, if not many, cases of early glaucoma?" Wilder, in an article written two years ago, made observation of this unfortunate fact and warned against it.

Equally frequent with the complaint of blurring, headache is what leads the patient to seek relief; and often sick headache or migrain from eyestrain seems an entirely satisfying diagnosis to make. But let me urge upon you, before accepting in your own mind such a diagnosis, to be sure and thrice sure that an attack of incipient glaucoma is not what the patient really has had, which almost perfectly fits the description of migrain that you are about to put down as your diagnosis; especially if the patient be over forty years of age, entirely over the attack when you see him, and has been relieved by "a good night's sleep."

Some years ago an article by a Canadian writer, showing records of case after case where field contraction was markedly present after migrain attacks, independent of the diagnosis of glaucoma, and with other progressive field defects, would lead one to believe that, after all, migrain is not merely the inconsequential malady we are apt to think it.

Accommodation inability without the factor of age or hyperopia should immediately arrest our attention. A comparatively young presbyope, who requires more reading addition than usual for his age, should always cause us to pause and think, especially if that need for increased reading addition has been repeated at shorter intervals than usual.

Examination of the anterior segment of the eye, first by daylight and then by oblique illumination and loupe, should be painstaking and thoro. We shall not more than mention the dilated anterior ciliary veins upon the porcelain white sclera, for the reason that ordinarily such would be found in more advanced cases than incipient glaucoma; altho, even in the noninflammatory form of the disease, when of long duration, this picture is often markedly present. Likewise the steamy cornea is ordinarily accompanied by enough deep circumcorneal injection to point the way for proper diagnosis by the oculist. Nevertheless a mild waterlogging of the cornea can be present with little or no congestive signs or dulling of the corneal surface, and it is only by oblique illumination that the slight haze is brought to view.

A shallow anterior chamber is so common as not to be taken particular notice of, except in association with a large pupil and particularly a relatively immobile pupil. A shallow chamber with a small, tight pupil is the usual finding in middle-aged people. But when such shallow chamber is met, associated with a large or wide pupil that reacts but little to light or accommodation in people of that age, it should immediately arrest our attention and suspicion. If, further, the iris has not the plump texture of health, is devoid of luster, and has the frayed out appearance of atrophy, with exposed pigmentary layer ring at the pupillary edge, glaucoma very likely has long been present even tho it may never have been manifested in the acute inflammatory form.

On examination with the ophthalmoscope, the media show us nothing abnormal between attacks, but the vessels, if carefully observed, do not match up rightly. The arteries are too slender in comparison with the veins, which are slightly distended and a little tortuous; not the ampulliform tortuosity we find in arteriosclerosis, but distended and tortuous thruout, indicating congestion, even tho but slight.

The nervehead now comes up for consideration; not the cavernous

atrophy of the optic nerve, which even the careless observer will readily see, but the cupped disc over which we stumble in differentiating between the socalled physiologic and the pathologic. Or, if an accepted pathologic cupping, the stage of cupping, whether of the preatrophy stage, or cupping with atrophy; the one in time, the other too late for help.

In considering cupping of the optic nerve, there are three types of cupping of the nervehead: 1, physiologic cupping; 2, atrophy cupping of ordinary optic nerve atrophy; and 3, glaucomatous cupping.

Physiologic cupping is partial cupping, never total cupping of the entire breadth, of the nervehead, nor deeper than the lamina cribrosa in its normal location. Any depth of the physiologic cup is due to its depth in the plump, heaped-up nerve fibers, and the vessels do not emerge at the very margin of the disc. The lamina cribrosa may be seen at the bottom of the cup, but not across the whole width of the disc at the floor of the cupping. On the other hand, a total cupping of the entire breadth of the nervehead from margin to margin is always pathologic, and must be either atrophy cupping or glaucomatous cupping.

The cupping of ordinary atrophy of the optic nerve is a shallow, flat, white, bleached out cupping, never deeper than the lamina cribrosa in its normal location, and the atrophy is present when the cupping takes place. In fact, the shrinking or atrophy of the nerve fibers makes all the cupping there is, with the fenestrae of the lamina exposed as a result of the shrinkage.

The cupping of glaucoma, rather shallow at first, is deeper than the normal location of the lamina cribrosa; is caused by its recession under intraocular pressure; and is there before atrophy of the nerve fibers takes place; in fact, the after-coming shrinkage of the nerve fibers in the already existing cupping adds considerably to its depth. At this stage we as oculists should recognize the cupping (recession of the lamina) before the shrinkage or atrophy takes place and while the nervehead is yet a healthy pink and the fibers plump; before the blue-green color appears, and before the shrinkage adds to the depth of the already existing excavation and the exposure of the fenestrae of the lamina. After these are present it is too late for help. At this stage, before the healthy pink color fades and before the fenestrae are exposed by shrinkage, the proximity of the vessels to the very edge of the disc should immediately put us on our guard. Later the characteristic bending over and disapearance of the vessels at the edge take place, but this latter is found after the added depth to the excavation resulting from shrinkage.

Even in the presence of a well defined glaucomatous change in the nervehead, in a hurried, superficial inspection, especially by indirect ophthalmoscopy, it is very easy to miss the tell-tale behavior of the vessels which, appearing at the bottom of the excavation, upon close examination can be seen to have a different hue, and by parallactic displacements can be seen to be at a marked difference in depth. This, I repeat, can be easily missed except in detailed inspection by direct ophthalmoscopy, especially where the nerve is still pink in health and before atrophy has arrived. With the added picture of contracted arteries and slightly distended veins, recognition at this stage is of vital importance, for just here, prior to atrophy, the picture does not include, as yet, a decrease in vision; and the time is here at hand, by proper medical and surgical relief, to arrest and avert the atrophy which does mean loss of vision. Even in the presence of large, unmistakable pathologic cupping, if the fibers are still pink, good vision and relatively uncontracted fields may be found. The above picture best applies to noninflammatory or simple glaucoma. But as time goes on, and especially since the advent of the tonometer, the noninflammatory is found not to be such an altogether different entity from the inflammatory form as it used to be considered.

Ordinarily, then, estimation of glaucomatous damage can best be made, not by the excavation but by the color of the disc (pink color of health or gray-white of atrophy) and by the size of the arteries. The halo around the

excavation is due to sclerosis and pressure atrophy of the choroid; but this, of course, is a late, too late, finding.

Field taking, I am sure, is something all of us shirk more or less, in the course of busy office practice, unless we have an assistant on whom we can depend. What man among us, with good training, does not experience frequently a pang of conscience when his eyes fall upon the old dust covered perimeter, and who does not feel the dread of an impending discovery of his negligence in permitting some case to slip thru his hands unrecognized, because of his shirking the task of field taking? Careful field taking certainly does take time and exhausting pains if expertly done, and calls for repetition of the task over and over again at frequent intervals in cases where it is of outstanding value, and the hardest for diagnosis. Fields should always be taken by the same operator and under the same conditions of light, personal control, and concentrated attention, to be of any value. Form fields are difficult enough, but color fields, the most necessary for differentiation in early glaucoma, call for intensified concentration of operator and patient, and a most exhausting patience and control on the part of the oculist. However, granting all this, to be worthy of our calling we must conform to the obligation, or smother our conscience and lay ourselves open to the chagrin of missing a diagnosis, that some more worthy confrere will, by his more conscientious self sacrifice, arrive at, and be rewarded.

In considering incipient glaucoma, we of course have in mind early glaucoma of all types, but all of us, I am sure, can recall cases of advanced disease which, so far as a careful history brings out, have never been manifested by even one definite inflammatory attack, and can be very properly classified as the simple or noninflammatory type; the treacherous, unsuspected form without outward sign to warn friends, associates, the family physician, or the oculist, of the insidious, slow but sure oncoming blindness, the thief in the night which is seldom recognized until the fading vision from atrophy is upon us and beyond any medical or surgical help. This is the form that most frequently slips thru our hands unrecognized, as refraction cases, and which we can only expect to catch by more thoro and painstaking examination.

As trained ophthalmologists in all our refraction work, we are duty bound to make careful and searching examination of the eyes of our patients to be worthy of the confidence and respect accorded us. Failing in this, our examination is not worth any more than that of the optometrist.

Fortunate, indeed, is the glaucoma patient who, early in the course of his affliction, suffers a decided inflammatory flare up, with all the distressing symptoms that accompany it; for he stands more chance, thru its early recognition, of being spared the long night of blindness.

The use of mydriatics and cycloplegics in patients in their thirties and forties for refraction is, in my opinion, not at all contraindicated as a general rule. In the first place, the oculist using cycloplegics in his refraction is generally the one who makes sure that there is no glaucomatous evidence to contraindicate their use. Then, a better opportunity is afforded for study of the fundus; and, finally, if there lurks an unsuspected tendency toward glaucoma, the use of the cycloplegic makes that tendency manifest itself, and often serves to put the patient, as well as the oculist, on guard for the rest of his life, where an insidious glaucoma might, coming on in later years, be not recognized.

The cycloplegic (homatropin at that age) given in the ideal way really cuts the danger of such procedure to a minimum, acting as a feeler; and can be counteracted much more surely than when given in the usual way. I refer to the use of homatropin over night, or the patient's dropping it into his eyes upon going to bed the evening prior to refraction the next morning. In this way any tendency toward tension has many hours in which to manifest itself, and the one drop thus given can far more surely be counteracted than the usual five or six drops put into the eyes five minutes apart at the office, as is the general custom. If in the morning the eyes are found to be entirely

free of any evidence of trouble from the instillation the night previous, three or four additional drops of homatropin can be given, and after a half hour wait retinoscopy can be done, followed by the trial case test. By this method far better and more complete cycloplegia will be found to be obtained.

In youth and young adults the instillation of one drop at supper time, bedtime, and breakfast time, followed by four to six drops five minutes apart at the office, will give such satisfactory and complete cycloplegia that atropin is seldom necessary. The annoyance of a week to ten days' disability at school for these young people is thus completely obviated, to the joy of all concerned. Try this method once, and you will always continue it, I am sure.

I have avoided reference to any pathology, medical treatment, or surgical procedure, in this paper, but it is interesting to notice in the literature the increasing and added reference to the circumlental space as the all important part of the eye in the origin of glaucomatous tension; also, in medical treatment, the reported startling effect of adrenalin subconjunctivally administered; and, in surgical procedure, for its relief, the favor in which iridotasis is held by many sound, conservative men.

In the development of surgery for glaucoma it would seem almost paradoxic that many of our most accepted operations have been the outgrowth of accidents in such surgery of the past. Mackenzie first observed that a puncture wound relieved a glaucomatous eye temporarily, and, as a result, paracentesis came into use. Later von Graefe discovered that a resection of part of the iris was beneficial to such cases, and came to do iridectomy as a regular procedure for its relief. It is not positively known how Lagrange came to realize the benefit of his sclerectomy operation; but it is likely the result, in the beginning, of a faulty corneal section in which a little of the sclera was included and which remained unattached under the healed flap of the conjunctiva.

Now we have iridotasis coming rapidly into favor. A few years back incarceration of any part of the iris in the wound would have deprived us of several nights' sleep; and it is likely that the observation of the relief and benefit following such an accident is responsible for its development. Most of us remember the articles written regarding the use of setons to promote subconjunctival seepage of aqueous fluid. Strands of silk, gold thread, and many other substances were proposed, but, of course, as foreign bodies they proved impossible. Now we have in iridotasis an autogenous seton, if you please; a strand or shred or tuck of the iris dragged into and intended to become incarcerated in the subconjunctival wound tissue of the operation. How strange that there should have proved to be virtue in our mistakes and a benefit from our accidents in surgery!

Likewise, to repeat, there may be considered good fortune in the inflammatory flareup from cycloplegia of a previously unsuspected noninflammatory glaucoma, for it is my belief that such, if it could be early recognized, would be just as amenable to medical and surgical treatment as the inflammatory form.

Let us all, then, resolve to be alert in the recognition of early glaucoma, and especially during the preatrophy stage, the stage where the recession of the lamina cribrosa is to be made out on careful inspection, where tho the cupping may be seen to reach from margin to margin, yet before the healthy pink color fades and before the atrophy coming on additionally deepens the excavation, and exposes the fenestrae in the lamina as a result of the shrinkage in the fiber bundles.

Failure of a trained ophthalmologist to recognize a frank case of acute inflammatory glaucoma is not to be thought of, but the early glaucoma, the incipient glaucoma, and the noninflammatory or simple glaucoma of even long duration, is the Waterloo of our specialty and should always be a conscious fear in the mind of every one of us, running hand in hand with the determination to short-cut no effort within the bounds of our ability to spare those trusting in our faithfulness from the progress of this dreadful affliction.

Lowry Building.

NOTES, CASES, INSTRUMENTS

SOLAR MACULAR INJURY.

HARRY VANDERBILT WURDEMANN, M.D.
SEATTLE, WASHINGTON.

CASE 1. Miss E. M. P., age 17. Consulted me Nov. 9, 1925, on account of a spot before the left eye. History: 3 years ago, in looking at an eclipse of the sun, without smoked glasses, she was temporarily blinded, but could see with the left eye until recently. Now she has vision R. = 6/8 +; L. = 6/LX; not improved by glasses. The fundus is normal in the right eye and there is no scotoma. In the left eye there is a central scotoma of 5°. The foveolas in both eyes are well defined and the crescentic reflex is present. The macular fibers are whitish. Wassermann and laboratory tests negative.

CASE 2. Miss R. P., age 20, sister. With the same history. There is a very small central scotoma in the right eye; the vision 6/LX direct, but full 6/6 when looking at edges of 6/6ths letters. The fundus is normal, except the macular fibers are pale. In the left eye the vision is 6/6; no defect of visual field, fundus normal.

Short notes on these two cases are herewith given because, in my previous experience, cases of macular disease or injury, following glare or solar eclipse or electric flashes, several years later have invariably shown changes in the macula resulting therefrom. Undoubtedly if the patients had been seen at the time of exposure, some tumefaction of the macula would have been observed. In these two cases it seems that only a few of the macular fibers of the optic nerve have been ultimately affected.

Cobb Bldg.

A CASE OF ELECTRIC CATARACT.

J. J. HORTON, B.S., B.A., M.D.
EAGLE PASS, TEXAS.

With the rapid increase in the use of electricity in industry the number of cases of cataract from electrical shock, to be reported, has also increased. Likewise, the belief that this type of cataract could be caused by electricity of high voltage only has been changed. I report the following case on account of the low voltage causing the lenticular opacity.

L. P., Mexican, age 21, coalminer. In the early part of May, 1925, while digging coal, an electric wire carrying a current of only 220 volts came in contact with the metal lampholder of his cap. He was knocked unconscious and remained so for about one hour. I examined his eyes as soon as he had received preliminary treatment for the shock.

Fig. 1. Case of electric cataract with burns of lid.

Above the right eye was a rather deep burn of the skin about 4 cm. in the horizontal by 2 cm. in the vertical directions. It extended from the junction of the outer and middle thirds of the brow to the medial edge of the opposite brow and included in its lower part some of the brow of the right side. Over the orbital ridge, below the middle of the lower lid, was a burn about 2 cm. in the horizontal and 1 cm. in the vertical directions and also quite deep. In the folds of the upper lid, medially, was also a small, but deep, burn. The skin surrounding these areas was red, and a thin exudate exuded from their surfaces. The conjunctiva was injected.

The next morning the palpebral and bulbar conjunctiva was moderately edematous and was injected. The patient stated that he could see when I held open the lids. There were no

signs of injury nor complaint on the part of the patient of the left eye.

On August 12th the company sent him to me for examination because he claimed not to be able to see with his right eye. Patient states that his vision has gradually grown less since the shock, until now he is unable to see anything except shadows with the right eye. He does not complain of the left. The scars from the burns are now well healed and reddish and occupy the positions noted above and shown in the accompanying picture. The one of the upper lid is small and triangular, but does not show up well on account of being in the loose tissues of the lids. The lids are otherwise normal and their movements normal.

O. D. Conjunctiva, cornea, and iris tissue appear to be normal. The pupil is round and of the same size as its fellow of the opposite side. It reacts a little sluggishly to light, but dilates fully and regularly with 1% atropin.

The aqueous is clear. There are many small roundish spots in the anterior capsule of the lens or in the cortex immediately beneath it. They are irregularly distributed. At first they appear gray, but upon careful illumination many are seen to be rusty. The posterior part of the lens cortex is flocculent and opaque. The peripheral part of the anterior cortex is less so. The central part of the anterior cortex seems to be liquid, and a single small, round pearl moves about in it with the movements of the head. Two thread like loops of greater density than the surrounding cortex subtend the periphery from 4-5 o'clock and from 5-6 and extend about halfway to the center of the lens. A third one extends from about 6-8 and two-thirds of the distance from the periphery to the center. The fundus reflex is very weak. Not even the disc or vessels can be seen. O. S. normal.

V. O. D. fingers at 2 ft. V. O. S. 6/6.

A needling was done on this date and another two weeks later. Altho the patient was progressing nicely, during my absence he was sent to another oculist. To quote his letter: "We did a cataract extraction and the patient had an uneventful recovery." "By the use of a heavy cylinder combined with a plus 10 sphere and prism 2 base in on the right his vision can be brought to 20/20 minus." Obviously extraction with resulting high astigmatism was unnecessary, as needlings and time would have been sufficient for complete absorption of the lens.

ACUTE CONICAL CORNEA.

FRANK A. MORRISON, M.D.

INDIANAPOLIS, IND.

J. T. S. was admitted to the hospital with a very indefinite history and only such as given by himself, no direct medical history being available.

The patient, a strong healthy looking young man of twenty-three years of age, stated, that in August, 1923, he had an attack of uremia, according to his physician, altho no examination had been made of his urine. Following this he suffered from "nervousness" with cramping of the arms and legs and vague bodily pains. His physician expressed the opinion that his trouble was due to his eyes and advised him to enter the hospital. He was kept in the hospital ten days under observation. The hospital examination, general, neurologic, laboratory and ocular was entirely negative and he was dismissed without a diagnosis altho hysteria was suspected. Three weeks later he again came under my observation saying that two weeks previous he had had another attack of "nervousness" followed by loss of vision in the left eye. There had been no pain whatever in the eye but on awakening one morning he discovered he could not see.

The cartridge like projection of the cornea was so extensive as to be easily seen thru the closed lids. There was no pain, redness or irritation of any kind. The right eye was absolutely normal. Having in mind the possibility of a like condition of the right eye it was gone over with extra care. The vision was 20/20, the corneal curvature was proven normal both by the shadow test and Placido disc. Tension with

the McLean tonometer 35. The left eye was operated upon in the usual manner by excision of the apex and sutures. Healing was uneventful. Ten days after this operation he had another attack of "nervousness" and immediately complained of loss of vision of the right eye. The right eye was now found in the same condition in which the left had been but rather more pronounced. It reminded one of a volcano with lava lines running down the sides. These lines stained deeply with fluorescein and conveyed the impression of cracks limited to the epithelium. The tension taken by the finger seemed normal. The operation of excision and suturing was likewise performed on this eye but was only partially successful and subsequently the galvanocautery was used. Ultimately both corneas became flattened and have remained so with a vision in each eye of approximately 20/80.

Willoughby Bldg.

LID AND FIXATION FORCEPS FOR TARSECTOMY AND GRATTAGE.

POLK RICHARDS, M.D.

FORT DEFIANCE, ARIZONA.

The advantage that I find in using the above illustrated forceps over other models on the market is that the lid, everted over the plate and clamped lightly, can be pulled up to any desired extent and fixed, thus permitting a good exposure of the retrotarsal fold. In tarsectomy, after the tarsus is removed, by loosing the adjusting end screw the tension is released which greatly facilitates approximation and the placing and tying of sutures. The instrument can be used with either general or block anesthesia on both the upper and lower lids. Canthotomy is a useful procedure when it is desired to expose the whole conjunctival surface including the culdesac with this forceps. Made with plates of two sizes, 20 and 25 mm.

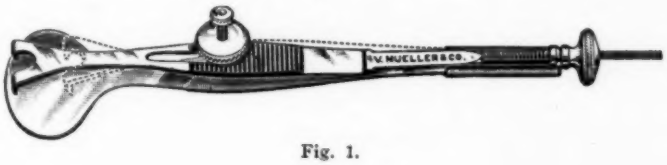

Fig. 1.

SOCIETY PROCEEDINGS

BALTIMORE MEDICAL SOCIETY.

Section on Ophthalmology.

March 25, 1926.

Dr. A. C. Woods, President.

Blue Sclerotics, Fragility of the Bones and Deafness.

Dr. Sinsky presented four cases of this well known syndrome. All were members of a single family. The family tree, which is shown in chart, is of particular interest in that it shows three generations of a family resulting from the marriage of two individuals, both of whom were said to have suffered from the triad. Only four members of the family had been examined. The data concerning the others was given by these four.

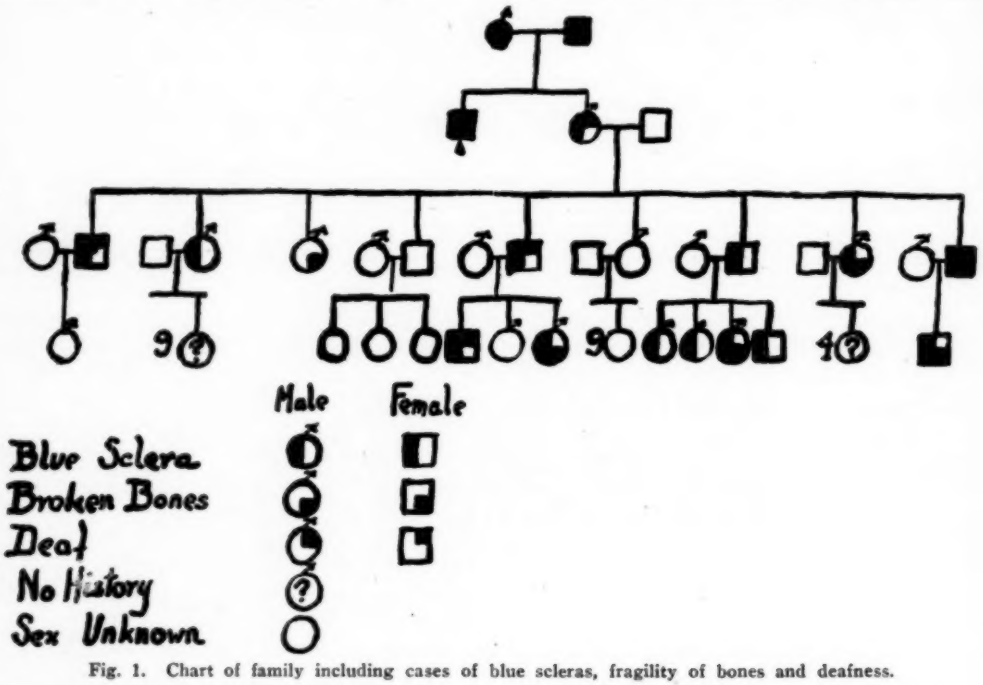

Fig. 1. Chart of family including cases of blue scleras, fragility of bones and deafness.

Color Sensitivity Tests.

Mr. A. E. O. Munsell of the Munsell Research Laboratory, presented a resumé of his studies which have been published in full elsewhere. The color of an object can be considered to vary in three different senses, namely, in brightness,—as from black to white,—in saturation,—as from brown to red,—and in hue,—as from red to yellow. Considering these three directions of variation, one can imagine a solid or figuratively speaking, a tree in which the color would vary from one point to the next in an even progression. The roots of the tree would be in utmost blackness, the highest tip would be brilliant white, the central trunk would be varying shades of gray and the limbs would represent varying degrees of color saturation from gray near the trunk to the most intense hues at their outmost tips. For commercial as well as scientific purposes, it is important to be able to standardize accurately the hue, saturation and brightness of different colors and it is with this aim in view that Mr. Munsell's research has been conducted. In the course of his investigations he has had occasion to test the color sensitivity of a considerable number of persons, with the interesting finding that there are quite enormous variations in color sensitivity among persons who by all ordinary tests are to be considered perfectly normal.

Sudden and Transient Blindness.

Dr. Harry Friedenwald read a paper on this subject, published elsewhere in this journal. (See p. 831).

Jonas S. Friedenwald,
Secretary.

THE MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Section of Ophthalmology.

July 13, 1926.

DR. JOHN J. SHEA, Presiding.

Cicatricial Ectropion of Both Eyes.

DR. E. C. ELLETT presented a patient, C. H. A., aged 59, who fell into a fire about March 1st and burned his face and head extensively and severely. When first seen on April 8th, there were still some unhealed spots on the scalp and over and under the left eye for which he was under the care of Dr. R. L. Sanders. Contraction of the scars was progressing, especially of the lids, having produced an ectropion of the inner half of the right lower lid and of the whole of the left lower lid. Operation on the left side was deferred until the burns had healed.

The right eye was operated on at once. Under local anesthesia the skin was incised down and in, parallel to the lid margin, and both lips of the incision were undermined and dissected up till the margin of the lid was brought up to its normal position, without tension. The lids were denuded at two places opposite each other and sewed together by mattress sutures, leaving the center free. A Thiersch graft was taken from the arm and put in the defect, and two sutures were placed to draw the lip upward, opening the raw surface to the fullest extent. The graft took and both points of suture of the lids formed adhesions, but one of them subsequently broke. The other still holds as a narrow cord, preventing any return of the ectropion.

The left eye was operated on April 16th, under local anesthesia, plus morphin and hyoscin. The same operation was done as on the right eye, with a longer lower incision and a similar one up and in. The grafts took but the lids did not unite, and the contraction of the lids continued, especially down and in, and up and out. On May 8th, a small corneal ulcer appeared on the left eye, from exposure of the cornea, and a second operation was done on this eye. The incision in the upper lid was made up and out, the lower one being placed down and in from the other graft. The lids were carefully sewed together. The grafts took and the lids adhered in one place. The cornea is now well covered and has given no more trouble. The grafts are all soft and flexible and resemble the rest of the skin very closely. The left eye waters a good deal, and the punctum cannot be located in either the upper or the lower lid. Vision, 20/30 and 20/40.

Discussion. DR. D. H. ANTHONY thought the important thing in this case was that operation was done early.

DR. P. M. LEWIS agreed that early operation was preferred. He follows the method of Wheeler, taking the graft from the upper lid.

DR. ELLETT had shown a boy before the society a year ago who had met with a similar injury and in whom operation was delayed until the contraction of the lids and exposure of the cornea which result had caused an ulceration of the cornea and practical loss of sight. In this case the point is, not that a satisfactory permanent result can be obtained by early operation, but that the eye can be protected and vision preserved. Subsequent operations will be necessary to get a good cosmetic result.

Intraocular Injury Necessitating Enucleation.

DR. M. B. SELIGSTEIN presented the case of Ivory Mukes, aged 9, admitted to the General Hospital June 29. He had been struck in the left eye by a piece of glass thrown by his brother June 28.

Examination showed large lacerated wound of cornea from 2 to 10 o'clock with marked prolapse of the iris. The right eye was not examined, but appeared normal on external inspection. On July 2nd, the left eye was enucleated under general anesthesia and a glass ball implanted. As soon as the child recovered from the anesthetic he complained of inability to see with the right eye. A fundus examination was immediately made and showed the disc to be much whiter than normal, and apparently atrophic. Careful history brought out the fact that following the injury to the left eye, he was unable to

see with the right eye. He was unable to see his way to the hospital and was led there. Wassermann was negative, and the pediatrician suggested possibility of juvenile tabes. At no time was there any inflammation in the right eye.

This case is presented to show how this might have been mistaken for a sympathetic ophthalmia.

Discussion. DR. ELLETT spoke of the necessity of always testing the vision in both eyes in injury cases as soon as possible, even in such slight injuries as foreign bodies in the cornea. Important questions of damage to the sight and large financial considerations are often settled by making such a record. He mentioned a case of foreign body in the vitreous resulting in the loss of the sight in the injured eye in which the test showed a defective vision in the uninjured eye. The records of the employer company showed that the man had good vision in the uninjured eye when he was employed and the result will be that the company will have to pay for that eye, tho it was defective before the injury in the other one, and was doubtless tested in a careless manner by some incompetent person.

DR. J. B. BLUE had had a similar case. The patient had suffered an injury of one eye. The other eye showed atrophic disc with choroiditis.

Bilateral Cataract Extraction.

DR. J. B. BLUE presented a case of Mrs. C., aged 47, who had been blind in the left eye for 25 years. Vision in right was very poor from same cause, namely cataract. Two weeks ago extraction of cataract with iridectomy was done on left eye. The following week similar operation on the right eye. Present condition showed both eyes healing kindly with media clear. Refraction was to have been done later.

Sclerocorneal Trephine for Secondary Glaucoma.

DR. J. B. BLUE presented a case of a patient who had stated that he could see well until May 10, 1926, when he developed pain and poor vision in left eye. This continued for a number of days, when the left eyeball ruptured with complete relief of pain. This eye was blind on admission to hospital. About May 20, 1926, the right eye began to have similar symptoms but less severe. Tension in right eye on admission very high. Vision: Could count fingers at 2 feet. The cornea above had a half moon shaped depression from loss of substance from former eye disease. The iris was adherent to cornea above. Since operation pain has been relieved, also the vision much improved.

The left eye was removed under local anesthesia, for staphyloma.

On account of very high tension, a posterior sclerotomy was done on the right eye two days previously.

Blood and other test negative.

Color Sensations of Uncertain Origin.

DR. ELLETT reported the case of W. S., a healthy young man, of 31, who was seen June 7, 1926. He had no eye trouble until last April, when he drove a car from Chicago, lost a good deal of sleep, and was exposed to wind, dust, and glare. Since then, he had had conjunctival symptoms, namely, a sandy feeling and some secretion. He also complains of a blur over the left eye, associated with the sensation of a larger blue spot in front of the eye. The vision was normal, the refraction under homatropin was O. D. + .50 sph. ⊂ + .25 ax. 90°; O. S. + .75 sph. ⊂ + .25 ax. 120°. The muscles, ocular movements, pupils and media were clear. The conjunctivae were slightly inflamed. Nose and throat were normal, tonsils not especially affected. He has had a fistulous abscess of one tooth, but this had been healed for a year, and gives no trouble. Examination with corneal microscope was negative. The light sense in the left eye was reduced. A smoked glass which reduced the illumination 50%, reduced the vision in O. D. to 20/40 plus, and that of O. S. to 20/80. He was put on strychnia. Three weeks later, he said he was about well of the symptoms. With the smoked glass he saw 20/40 in each eye.

The only suggestion that was offered to explain this case is that of the effects of glare on the left eye. In driving a car, the left eye is on the open side, and the glare from the road might have affected it so that the afterimage of the yellow of the road, namely, blue, its complementary color, was annoying for a long time.

P. M. LEWIS,
Secretary.

American Journal of Ophthalmology

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OPHTHALMOLOGY OR OPTOMETRY.

Medicine, the healing art, not a drug or the giving of drugs, reaches into every domain of science for new means to maintain and restore health, or to combat and overcome disease. Physics, chemistry, botany, zoology, bacteriology, psychology, all are laid under tribute to promote the health of man and the animals that he uses and protects. Every resource of civilization, that can be applied to meet health needs of mankind, is to be used in the healing art, by workers most competent to use it.

When the sight testing opticians called themselves optometrists, their most shrewd and farsighted leader gave them the slogan: "The optician treats light—the oculist treats disease." This was very popular with those who were trying to win professional status, by legislation and advertising; and was effective with newspaper reporters and politicians. But no optometrist has been shrewd enough and farsighted enough to point out when light needed treatment, except as it lowered the powers or impaired the health of the human body. Light that causes symptoms, whether photophobia, faint or blurred retinal images, headaches, nausea, vertigo, or squint, needs treatment. For the sufferer from symptoms, produced by light or anything else, the best thing to do is to go to the physician for expert advice, as to what there is in his environment or habits of living that is causing his trouble, and have that "treated" in the way that will give relief.

When Donders and Helmholtz called attention to the importance of physiologic optics and the pathologic influence of eyestrain from errors of refraction, it at once became a part of the business of physicians who gave attention to eye defects and diseases to prevent and relieve such symptoms, by prescribing lenses or prisms as they would prescribe other mechanical remedies, or operations, or drugs. The making of lenses and fitting them to the face had before been done by opticians; and some of these sought to add the prescribing of glasses to their business; and by legal requirements and restrictions to make that business a profession which they called optometry.

The right prescribing of glasses requires two things: First, a knowledge of medicine to decide if headache, vertigo, nervous dyspepsia, poor vision, anemia, or other pathologic conditions come from eyestrain, eye disease,

sedentary occupation, chronic infection, organic disease of the brain, inflammation of the nasal accessory sinuses, or other disease. Second, the knowledge and patience needed to measure accurately the refraction of the eye, to determine what glasses, if any will remove the eyestrain, or give the best vision obtainable by that particular pair of eyes the patient happens to have.

Before Donders and Helmholtz, very few physicians had any practical acquaintance with optics, and very few opticians had any practical acquaintance with the diagnosis of disease. Since that time a great deal of energy on both sides has been expended in claiming, for each, that the prescribing of lenses was his own prerogative, and in keeping the other party from doing it, without fully preparing themselves to perform this function to the best advantage of the community. Medical schools have given some instruction in optics, but not nearly enough to fit physicians to care for the optical needs of the community; and optometrists have tried to learn a little about the eye and its diseases, but were generally content with only such a smattering, as might enable them to pretend medical learning.

But these attempts have not settled, and will not settle, who should prescribe glasses. That will be decided only when medical schools give, and physicians take, adequate instruction in optics, in relation to physiology and pathology; or when optometrists understand that they can build up a profession similar to that of dentistry, only if they include in their training for it, the fundamental branches of a medical education. On both sides there have been individuals who saw that the needs of modern students included more ophthalmic study and teaching than had yet been offered for them. But physicians who knew nothing of optics have dominated the medical schools; and physicists and mechanics who knew nothing of pathology and diagnosis have controlled the teaching, and legislative panaceas have secured the largest following among the optometrists.

The struggle for dominance in teaching or political action, will not do much to improve the health or the usefulness of the vision of the nation. Competition in thoro teaching of what practitioners need to know is needed to develop the efficiency of all who undertake to advise about glasses, or the care of the eyes. For oculists the more serious and general study of optics, with careful training in the accurate measurement of refraction is needed, to ensure their efficient service to the community; and to secure the respect and confidence for the medical profession that is the surest protection against the devastating quackery of short-cut eyesight specialists. Almost 15 years ago, an optometrist wrote: "Our right to exist is based on the fact that oculists, as a rule, do not qualify in optics, and not upon the assumption that optometrists do not need to qualify in medicine."

Very slowly and imperfectly have these defects in training been removed. The claim that every physician is fitted to prescribe glasses, betrays a low estimate of the qualifications needed for that branch of ophthalmology. The proposition to give every "general practitioner" a smattering of optics, that would enable him to compete with the average optometrist, shows as poor an understanding of the needed qualifications. Both betray ignorance of what the exact correction of refraction has done for some chronic sufferers; and of the greater number of others who will be helped by similar treatment in the future. To assume that the diagnosis of eyestrain is a simple and easy matter for which the student is fitted by his undergraduate studies, is little better than to assume that any optician who could sell spectacles to a presbyope or a myope that would clear his sight, was fitted to treat headache, vertigo and epilepsy, not to mention diabetes and tuberculosis that have been ascribed to eyestrain.

We need not be greatly concerned about the defective training of optometrists. The failure that will come to them from too much faith in legislation and plausible advertising, will not hide the results of ignorance and lack

of training in a profession that might with greater reason be expected to meet all the health needs of the people. The medical profession must be prepared to attack problems in optics with the same intelligence and industry as problems in chemistry, or bacteriology. If the oculist will adopt standards for achievement as high as those of the old fashioned clinician, or the aseptic surgeon, it will matter very little what claims the optometrist may make, or how he may advertise them.

It must be frankly admitted that the undergraduate course in medicine can not make a finished specialist in ophthalmology or in any other branch of practice. The physician, or surgeon, who achieves success in special practime must do it by graduate study. The opportunities for such study become, every year, wider and more accessible. Earnest, persistent effort in that direction will make the ophthalmologist successful in practice, respected in his profession, worthy of the high esteem of all honest workers and the gratitude of the growing circle of those relieved by his skilled and loyal service. To furnish better graduate teaching in refraction, to make it more accessible to those interested in it, to urge the importance of this work on the medical profession and the public, are duties that rest on all who appreciate the importance of good vision to the workers of our time, and the difficulties that have to be overcome to help people to see most clearly and most easily.

E. J.

TRACHOMA IN RUSSIA.

The dramatic promoter of an eye water, Colonel Sellers, used to say on the stage: "There are 500,000,000 people in Asia and they all have sore eyes." In an article by Major W. R. Dear, M.C., U.S.A., formerly physician in charge of the Kazan District of the American Relief Administration Operations on the Volga in Russia, elsewhere abstracted, in this JOURNAL (p. 789), the terrible hygienic conditions of the Russian peasants and the absence of medical services due to the destruction of the "Intelligensia" by the "Bolsheviki," is dramatically described.

While the chaotic conditions of government in Russia obtain, it will be impossible for liberal minded and charitably disposed Americans to do anything for their immediate relief. We may, however, in the future look forward to the carrying of this contagion from Russian to contiguous territory, and thence by emigrants even to America, perhaps bringing in a more virulent form than the easily treated and curable type of trachoma that is now endemic in spots in the United States.

When we hear that 75 per cent of the population of the Chouvash Republic in 1922 had trachoma; that 8 to 10 per cent are incapacitated from work because of partial blindness, and that many of the cases of blindness seen in Russia, at least 22 per cent, are blind as a result of trachoma, it seems to show that this is indeed a serious condition which merits our notice at this time.

The old Russian government kept up ophthalmic hospitals and equipped physicians for ophthalmologic practice; but now death has overtaken those who knew how to combat the disease, and the conditions are for the present hopeless. The civilization of the rest of the world, in this enlightened Twentieth Century, has developed altruistic and humanitarian elements, and will sooner or later have to do something; not only to clean out this pest hole of Asiatic influence, but also to prevent the rest of the world from becoming blinded by trachoma. In the meantime the most strict examination of immigrants by the authorities should be kept up; and in addition medical surveillance of the few people who are permitted to come in from Southern Europe should be instituted.

H. V. W.

The account by Major W. R. Dear in "The Military Surgeon" for June, 1926, of the trachoma problem in certain parts of Russia reveals an appalling situation. No accurate figures are available since the recall of the American Relief Administration in 1923, but there is every reason to believe that conditions have grown progressively worse. In one area in a population of about one million, approximately 75% were afflicted.

In Russia trachoma is by far the chief cause of blindness, being responsible for twenty two percent as compared with less than two percent in Western Europe.

In 1914 there were only 209 ophthalmologists to care for the one hundred and twenty million population; and now there are even less to attend to these people impoverished by war and weakened by poor living and, therefore, an easy prey to diseases which thrive on squalor and malnutrition. An effort was made by Prof. Chirkorski to found a trachoma institute in Kazan, sponsored by Americans some years ago, but its present existence even is doubted by Major Dear.

Our government is doing a splendid work in the trachoma field thru its Public Health department, especially at the trachoma hospitals recently established in our country. The best methods of spreading instruction in prophylaxis and treatment are being studied, and the dissemination of this knowledge is probably the most important part of the work. A great many cases are alsb aided by the treatment given at the trachoma centers, and in tours of physicians thru the infected areas. Research is being carried on in an ever broadening way towards the discovering of the etiology of the disease.

Political unrest in the Soviet Republic has hampered the formation of such organizations as our National Comittee for the Prevention of Blindness which might direct an organized attack upon trachoma. It is probably impossible at the present time to aid in any national official way, but this does not preclude the possibility of something being done by individual effort.

L. T. P.

BOOK NOTICES.

Contributions to Ophthalmic Science. By American Authors. Quarto, Cloth, 340 pages, 127 illustrations, 22 plates. Menasha, Wisconsin. George Banta Publishing Company, 1926.

The desire on the part of pupils and colleagues in the United States, to honor Dr. Edward Jackson by a special token of appreciation on the occasion of his seventieth birthday, found expression in the publication of a volume bearing the inscription: "Contributions to Ophthalmic Science—dedicated to Dr. Edward Jackson in honor of his seventieth birthday, March 30th, 1926, by pupils and colleagues in the United States."

Bound in maroon linen with blind stamping and lettering of gold, the small quarto presents a very attractive appearance. The text is cleanly printed in bold round faced type on light paper of good quality, coated paper being used for the numerous and excellent illustrations in black and white, and colors.

The volume contains three hundred and nineteen pages. There is an editorial note by the Committee on Publication; an appreciation of Dr. Jackson by Dr. George E. deSchweinitz; a complete bibliography of Dr. Jackson's contributions to ophthalmology and general medicine by Miss Mabel Dean Anderson; and a list of subscribers. A satisfying portrait of Dr. Jackson forms the frontispiece. Two hundred and ninety-seven pages are devoted to the forty contributions forming the body of the volume. In passing one must note with great regret that time has already taken toll of four of the contributors—Doctors Duane, Feingold, Francis, and Ziegler. It is impossible here to review these papers in detail; but reference to their main points may be of service, in view of the difficulty that is at times experienced in gaining access to works of this kind.

In Marcus Feingold's case of "Abnormal Movement of Upper Lid, Rising with Downward Gaze" the phenomenon is attributed to jumping over of the impulse to innervation on to the adjoining ganglion cells of the levator palpebrae. Lewis Ziegler's contribution is a comprehensive study of "The Problem of Choroidal Hemorrhage in Cataract Extraction," with personal observations, instructive conclusions, and a complete bibliography. Under "Massive Gliosis of the Retina" Jonas Friedenwald describes and illustrates a benign retinal tumor of highly differ-

entiated glial tissue, and a hitherto undescribed type of glia cells normally found in the optic disc. Phinizy Calhoun ascribes the "Cyanosis Retinae occurring in a Case of Erythromelalgia which Terminated in Raynaud's Disease" to lack of vasomotor tone. Dr. Alexander Duane's "The Norms of Convergence" is a masterly study of the normal converging power that will stand as a classic. Under "Some Improvements in Histological Technic." F. H. Verhoeff gives valuable suggestions in regard to keeping the retina in position, opening the eyeball, decalcification of tissues, staining with eosin, and the preservation of hardened tissues and unmounted sections. Albert C. Snell and Scott Sterling present the data, deductions, and conclusions of experiments undertaken to determine the percentage relation between all graduations of macular visual acuity, as expressed and measured by the Snellen formula, and macular perception, i.e., the physiologic function of the macula. Harold Gifford describes unusual clinical features of glioma of the retina and advances therapeutic suggestions. Arnold Knapp points out the value of X-ray photographs of the optic foramen in determining intracranial extensions of optic nerve tumors, and comments upon the operative difficulties in cases of this sort. Manuel Uribe-Troncoso reports additional cases of gonioscopic examinations in glaucoma. Shahan writes of his work on the "Thermal Death Points" for normal and neoplastic tissues; and McReynolds refers to "Some of the More Difficult Problems in the Management of Pterygia" arising in his personal practice. Berens, Hardy, and Pierce give the results of their studies in ocular fatigue with a new type of ergograph that they regard as of indubitable value in diagnosing and treating certain types of ocular fatigue in aviators, and as an adjunct to the equipment of the ophthalmologist. Lee M. Francis's "Unusual Congenital Anomaly of the Crystalline Lens (Atypical Anterior Lenticonus)" was thought to be due chiefly to a defect in the nucleus or perinuclear layers. William Zentmayer and Calvin O. Rush report and summarise the findings in eleven cases of nodular degeneration of the cornea (colored plate). The cause of Edward H. Cary's case of congenital total ophthalmoplegia was conceived to have been a nondevelopment of the more rostral portion of the general somatic efferent cell column. Lloyd Mills advocates the use of the sclero-conjunctival flap in cases of congenital and juvenile cataracts. Emory Hill presents observations based on an analysis of thirty-five cases of papilledema; and L. G. Wesson, E. I. Burky, and Alan C. Woods report their results with several new preparations of uveal pigment suspensions.

The title of William T. Shoemaker's paper "Serous Exudate beneath the Retina Clinically Stimulating Sarcoma of the Choroid or Ciliary Body" fully covers the contribution. A pathologic report on the enucleated eyeball, by Perce De Long, accompanies the report. Arthur J. Bedell's communication records examples of two groups of unusual anterior capsular changes, examined by the slit lamp: the one showing changes anterior to the intact capsule; the other rupture of the anterior layer of the capsule. Harry Friedenwald reports two cases of sudden unilateral blindness after middle life, which were of special interest in that, in both, the onset of the blindness was coincident with serious cerebral lesions. Robert Von der Heydt's paper has to do with aqueous currents, and cells and cellular deposits in the anterior chamber.

Luther C. Peter gives a resumé of the literature dealing with the medical treatment of incipient cataract in adult life, and treats of the therapeutic measures which he has found of value in his own practice. W. H. Crisp describes a rare form of membranous cataract. James M. Patton treates of metastatic suppurative choroiditis and panophthalmitis and the ocular evidences of thombosis of the cavernous sinus; and Walter R. Parker enters fully into the pathology of a case of "Concretion in the Canaliculus" (colored plate). Meyer Wiener gives the results of his work in the surgical removal of corneal opacities. Frank

Burch reports on a case of coloboma of the lid. L. Webster Fox outlines our present-day treatment of senile cataract; and George Slocum presents a detailed description of a unique case of retinitis striata.

Martin Cohen, John A. Killian, and Mannette Metzger report on their investigations, the ultimate object of which was to determine whether the development of primary glaucoma is associated with any significant change in the chemical composition of the humors, or of the lens of the eye, and whether such changes are dependent upon analagous changes in the blood or other body fluids, in the relation of cause and effect. The use of the suture in cataract operations is fully dealt with by E. C. Ellett. Two cases of tendon transplantation of ocular muscles are described by W. S. Franklin and F. C. Cordes. S. R. Gifford reports on multiple myxoma of the orbit; and Harry S. Gradle treats fully of lens opacities in diabetic patients.

H. V. Würdemann's contribution is really a splendid atlas of ophthalmoscopic changes in the macula lutea (fifty-four pictures painted by the author himself), the subject matter being largely descriptive of the accompanying illustrations. H. Maxwell Langdon covers comprehensively the subject of "Ring Scotoma"; and Everett L. Goar that of "Primary Endothelioma of the Optic Nerve Sheath." William C. Finnoff closes the series with a thoro pathologic study of a case of "Traumatic Internal Cyclodialysis and Siderosis of the Eye."

The contributions without exception are of a very high order, clearly and succinctly written,—entirely worthy of the authors, and of the distinguished and learned teacher, writer, investigator, and practitioner whom they delighted to honor.

W. Gordon M. Byers.

Physical Therapy in Diseases of the Eye, Ear, Nose and Throat. By **Abraham R. Hollender, M.D.**, and **Maurice H. Cottle, M. D.**, Chicago. Bound, 307 pages, 81 illustrations, 7 tables. Published by The Macmillan Co.

So far as the reviewer knows, this is the first book published dealing exclusively with physical therapy of the eye, ear, nose and throat—at any rate, it brings together in a compact form what information there is on this subject. The book is divided into six parts, with an appendix. Part I is a discussion of electrophysical agents and the apparatus pertaining to them—the galvanic current; high frequency currents —diathermy; the sinusoidal current; radiant heat-light; ultraviolet light; X-rays, in all, 78 pages. Part II, 31 pages, describes the treatment of diseases of the eye. For a statement of the various conditions in which physical therapy is indicated, the reader is referred to the book itself. The authors quite rightly note that the usual medical treatment must be carried out in addition to the physical therapy. A mistake on page 96 credits Shanahan instead of Shahan with the discovery of the thermaphore. 44 pages are devoted to the ear in part III. Part IV, 30 pages, takes up diseases of the nose and accessory sinuses. Part V, 40 pages, treats of diseases of the mouth, pharynx and larynx, while Part VI, 52 pages, deals with border line conditions. The appendix gives the results of certain experiments performed on animals. At the end of each Part, there is a bibliography, and there is an efficient index. The paper and printing are good, and the book is of a convenient size for the library, and a valuable addition thereto.

C. L.

The outstanding feature of this book is the description of different forms of apparatus and the manner of their use. It has been written by otolaryngologists, but Hollender is also the editor of the Archives of Physical Therapy. It contains an explanation of the electrophysical agents and their application which will be especially helpful to the man who is well grounded in his specialty but has not counted these agents among his therapeutic resources. The Part on Diseases of the Eye seems to be based on the general experience of most oculists who have tried these measures. It is certainly better to get one's impression of this branch of therapy from a book of this

kind, than from the claims put out in support of particular makes of apparatus.

E. J.

Otogenic Paralysis of the Abducens. Wm. Hardin Sears, M.D., F.A.C.S., Huntington, Pa. Paper, 8vo., 66 pages, 7 illustrations. Reprint from Trans. Amer. Laryngological, Rhinological and Otological Society, 1925.

This was written as a candidate's thesis and contains a report of 26 cases, heretofore unpublished, including 2 of the writer's own; a tabulation of 172 cases, 145 previously recorded in the literature; and a bibliography of all references recorded to date. It makes especial mention of isolated palsy associated with irritation of the Gasserian ganglion. The illustrations bring out the anatomy of the parts, considered with reference to the possible routes of infection.

Abducens paralysis should always arrest the attention, and become a matter of great interest to the oculist. The main facts regarding the 172 reported cases are presented in a table. The cases here published for the first time are reported in detail. This monograph with the bibliography that accompanies it, will be a valuable work of reference, for all who have to do with this serious complication of otogenous disease.

E. J.

Tenth Annual Report of the Ophthalmic Section, 1922. Ministry of the Interior, Department of Public Health, Egypt. Paper, 48 pages. Published by the Government Press, Cairo, 1926.

This is a report, chiefly in tabular form, of ophthalmic conditions in Egypt, with special reference to Cairo. Probably the most interesting sections are those devoted to the Statistics of Blindness and Statistics of School Clinics. In 1917, 12 per 1000 of population were blind. The chief cause was acquired conjunctivitis, resulting in lesions of the eyeball. This is especially interesting in view of the statement that there is very little secondary infection following glaucoma operation, one of the reasons for this latter fact being the more "resistent nature of the conjunctiva of the Egyptian." The Hospital Statistics show that there were almost twice as many patients treated in 1922 as in 1919, chiefly outpatients, as the number of hospitals had not increased. The number of times that the gonococcus was found was 11305, being more than all the rest together. The next most frequent organism was the Koch-Weeks bacillus, 5421. The trachoma statistics of school children are appalling! In only one school was there as low as 60%; the highest was 99.1%; average 89.8%. There is a large amount of additional, valuable information contained in these tables, which would more than repay the investigator. The book may be obtained from the Government Publication office, Ministry of Finance, Dawawin P. O., Cairo, Egypt.

C. L.

Legalized Optometry and the Memoirs of Its Founder. Charles F. Prentice, M.E., Physical Eye Specialist, New York. Memorial Edition. Cloth, 12mo., 416 pages, 4 illustrations. Seattle, Casperin Fletcher Press, 1926.

The preface to the first part, on Legalized Optometry, signed by the publishers (having the same initials as the author) sets forth its intention "to narrate the history of optometry legislation in the United States of America." Further, it points out "the world at large is not informed concerning the reason for such legislation." Also "it is hoped that ophthalmologists will accept this recital of the truth without prejudice and with full preparedness to make righteous allowance for the provocation that justified the optician's acrimonious arguments." The edition "is limited to 200 copies for distribution among select subscribers."

The work of Donders, Helmholtz, Knapp and others fixed the attention of many physicians on the importance of ocular refrection thru its influence on human physiology and pathology. The medical profession in the United States was seeking by medical practice

laws to draw the line between the educated physician and the ignorant claimant to medical skill. Some who appreciated the importance of refraction, attempted to include the prescribing of spectacles in the legal definition of medical practice and only to be done by the regularly educated and licensed physicians. Before this glasses had been made and sold by opticians. Physicians had rarely concerned themselves about the possible needs of patients in that direction; and very few of them knew anything about optics or ophthalmic lenses.

Among the opticians of New York was James Prentice, who had been a maker of drawing instruments. He came from London in 1842, established himself in that line of business and about 20 years later had opened a retail optical store. His son Charles F. Prentice, was educated in Germany as a Mechanical Engineer, joined his father in the optical business and succeeded to it at the father's death. Prentice in 1884 to 1890 published some important papers; describing a class model for demonstrating refraction and accommodation; a treatise on ophthalmic lenses; and papers on dioptric formulas for combined cylindrical lenses; and on the metric system of numbering prisms (the prism dioptry).

In 1892, physicians prescribing spectacles chose to send their patients to certain opticians to be supplied with lenses. Prentice found his business interfered with and was informed from Dr. Roosa, Chairman of the Committee on Medical Legislation that "if he wished to advise people about glasses, instead of merely selling them on their order, or that of the oculist, he must first get a degree of M.D. and then a license to practice in this State."

He at once appealed to opticians to organize and seek legislation to protect them in their established business; and in 1896 a bill to enable the Optical Society of the State of New York to regulate the practice of opticians, was brought before the Legislature. This bill was defeated, but the struggle was continued until in 1908 the optometry law was passed, creating a state board of Examiners in Optometry, under the authority of the Regents of the University of the State of New York. Optometry laws had already been passed in 12 other states.

Prentice was appointed on the Board of Examiners in Optometry, became its President and during ten years was active in its administration and defense. During this period he suggested and brought about the establishment in 1910, of a course on Optometry in Columbia University open to students, medical and nonmedical. The history of this period appears largely in the "Memoirs," 228 pages, which constitute the second part of this volume. With it is told the history of a struggle among the optometrists as to whether the optometrist should assume the title of "doctor" or seek it from the trade schools which offered it. Columbia University did not offer any such title to its graduates in optometry. Prentice consistently opposed it, preferring to be called Mister and suggesting the titles "Optocist" and "Physical Eye Specialist." He understood the significance and value of the Academic title of Doctor, and would take nothing of the kind that he had not really earned. However, there were many optometrists that did not understand academic distinctions and were willing to seek a title they believed would add to their prestige and financial returns.

At the Buffalo meeting of the Optical Society of the State of New York in 1912, Prentice clearly stated his views using this illustration "Lunatics do not know or admit that they are crazy, and optometrists who fail to appreciate the principle upon which academic degrees are acquired in New York, similarly do not realize their own folly in calling themselves doctors." The convention applauded him but later elected as President a man who "affirmed that the Society must understand that he would, regardless of protest, assume the office as Dr. X." Prentice resigned from the Society. In his Buffalo address he also said "I have for years compared optometry with dentistry, on belief that "all future practitioners of optometry should be graduate physicians specifically in eye practice, because, universally, physi-

cians do not qualify in optics, optometry or ophthalmology." Furthermore, he said "There is not an optometrist in the land who is sufficiently versed in medicine, either by collegiate education or amateur experience, to prescribe the required scope of medical education that should be given to the more advanced practitioner of optometry." This is the basis on which Prentice continued to "strive for the logical uplift of optometry." His willingness to admit that the "eye practitioner" should know medicine as well as optics should make his memoirs of interest to ophthalmologists.

There is more in them which is interesting. The writer of this autobiography uses the opportunity to give expression to a wide range of reflections and observations, that should prove interesting to a wide circle of his contemporaries. In a dozen pages he points out how parental influence develops character. He describes his personal experience of the medicinal virtues of Scotch whisky in small doses to help in convalescence. He states that fortunately he "never became a gluttonous Bryan or a bibulous Bacchus." But in two widely separated sections, pages 202 and 386, he digresses from optometry to set forth at length and with emphasis his views of prohibition and the philosophy of human nature and the province of legal enactment that supports it. His book is of interest, too, because of the glimpses it gives of men and movements at the end of the ninteenth century. "Optometrists," ophthalmologists, politicians, statesmen, job holders, physicists and educators, figure in it. We hope that copies will in time find their way into all medical libraries.

E. J.

Cannula Implants and Review of Implantation Technic in Esthetic Surgery, by **Charles Conrad Miller, M.D.,** bound, 99 pages, 11 illustrations. Published by The Oak Press, Chicago, 1926.

This book is essentially a review of the author's experiences in hypodermic implantation for cosmetic purposes, with a recommendation of the use of small bits of gutta percha introduced beneath the skin by means of a cannula. About 50 pages are devoted to a translation of a paper by Prof. S. Bourack, Ztsch. f. Nasen. u. Ohrenheilkunde, 1925, p. 441, the relevance of which is not exactly clear. The sources, different varieties and methods of preparation of gutta percha are gone into at some length, and the remainder of the book is a rather rambling discussion of various forms of implants, with a description of the author's technic of gutta percha implants. The illustrations are exclusively of the cannula and trocar, and of the method of preparing various kinds of rubber for use in the cannula. It is hardly likely that this procedure will be of much use to the ophthalmologist, especially in view of the last sentence in the book: "For the present patients should be told that these methods are not as yet recognized as established surgical procedures."

C. L.

Eye Sight Conservation. Lantern Slides and Lecture Material. Paper, octavo, 96 pages, illustrated. The Eye Sight Conservation Council of America. New York City.

This, Bulletin 5, of the Eye Sight Conservation Council of America, gives information of the material that has been prepared and accumulated to assist lecturers who will undertake to spread the gospel of: "Defective vision and its correction, eye protection and proper illumination are of vital importance in our schools and colleges, in our homes, in industry and in all walks of life."

Among the officers and directors of this organization there is no physician; and on its "Board of councillors," which numbers 10, there are but 2 who have received the medical degree; one of these is a State Health Officer, and the other the head of a teachers college. We learn: "The management in its appeal for funds encourages support from firms and individuals with a business or professional interest in optics, eye protection and lighting."

From an examination of the material thus brought together, it would appear

that the lectures based on it and illustrated by it, would chiefly promulgate the importance of "Eye Sight Conservation" by wider use of ophthalmic lenses, protective glasses and improved systems of lighting. That those who have "business or professional interests" in this direction should seek to promote them by propaganda is not surprising. If spectacles and new lighting fixtures receive undue emphasis as aids to such conservation, it may be the duty of the medical profession to furnish a better and more complete program of health appreciation and emphasis; either thru the Committee for the Prevention of Blindness, an organization quite different from the Eye Sight Conservation Council, or in such other ways as it may devise and institute.

Meanwhile, some oculists may find available in the lantern slides and hints illustrated and suggested in this pamphlet, valuable assistance for the preparations of popular lectures on ocular hygiene.

E. J.

United Fruit Company Medical Department. Annual Report 1925. W. E. Deeks, M.D. Paper, small quarto, 320 pages, illustrated. Boston, United Fruit Co., 1926.

Nearly 100 pages of this report deal with organization and statistics. The number of patients in the 8 hospitals of the Company in Cuba, Columbia, Central America and Mexico averaged 1064 for the period covered by the report. The proportion of these cases in which the eye or its adnexa were under treatment is small, about 1 per cent in hospital cases and 2 per cent of the dispensary treatments. Only a few of the diseases of the eye are specified, follicular conjunctivitis and trachoma being the most common. The total number of cases of eye diseases was 3,105. Among eye operations, by far the most frequent was removal of foreign body, 386. After this came enucleation and incision of abscess, each 14, and extraction of cataract, 9. Among more than 30 original papers, the only one approaching ophthalmic interest is one on Functional Disorders Attributable to Tropical Sunlight. This will be abstracted elsewhere.

Some of the general diseases as malaria and amebic dysentery have important eye symptoms but these are not considered in the papers that treat of them; which deal rather with causation, frequency, distribution and drug therapeutics. These aspects of general diseases are important even if one confines his practice to a narrow field of special diseases; and this volume furnishes information about them that is practical and quite up to date.

There are few current publications on medicine that are teaching so much of the pathology of tropical diseases. This causes no surprise when it is noticed that special laboratory workers are serving in most of the company hospitals, and that among their consultants are such men as Mallory and Rosenau of Boston, Banting of Toronto, James of the University of Virginia and Castellani of Florence, Italy.

The service on which this report is based makes it practical, and the scientific work done in connection with it gives it permanent value in medical literature.

E. J.

CORRESPONDENCE

Proper Credit.

To the Editor: In the Abstracts of your April issue, page 308, you deal with a case of "Epithelioma of the Orbit in a Young Boy," which was published in my annual report for 1924. The authorship is erroneously attributed to Zachariah, G., whereas I am responsible for the notes and pathologic findings referred to.

Yours faithfully,

Madras, India. ROBERT E. WRIGHT.

[The Editor must apologize for the error for which he is responsible, altho to avoid misstatements is often difficult. The Report of the Government Ophthalmic Hospital at Madras is a Government Report, not prepared primarily to promulgate scientific observations and assign the proper credit to authors. However, these reports of cases are so good, they should be noticed, even at the risk of miscrediting their authorship.]

ABSTRACT DEPARTMENT

Reprints and journal articles to be abstracted should be sent to Dr. Lawrence T. Post, 520 Metropolitan Building, St. Louis, Mo. Only important papers will be used in this department, others of interest will be noticed in the Ophthalmic Year Book.

Troncoso, M. Uribe. Anatomic Notation of the Visual Field. Brit. J. Ophth., 1926, May, v. 10, no. 5.

This is a plea for the adoption of the anatomic notation of the visual field in place of the present confusing method, thus avoiding the technical difficulty one encounters in twice inverting the mapped findings of the perimeter as usually charted. The present method originated in the use of the blackboard and campimeter in which the examiner stood behind his patient. With the advent of the perimeter the examiner faces his patient thus causing a readjustment of the mental picture. In the anatomic method, the mental process of inverting is avoided by making notations perforating the chart and tracing on the reverse side of the present charts. Thus the records of the fields will be in the anatomic position. The notation of the meridians in the charts should be standardized similarly to the axis of the cylinders in a trial frame, that is 0° always at the left of each eye; 180° to the right and 90° above, 270° below. Three illustrations accompany the contribution.

D. F. H.

Obarrio, J. Is the Diplopia in Epidemic Encephalitis Always Paralytic? Klin. M. f. Augenh., 1925, Sept.-Oct., 75, p. 455.

A boy, aged 7, suffering from epidemic encephalitis lethargica, showed horizontal diplopia when wakened. It disappeared after he was entirely awake and did not recur excepting when he again fell asleep and was wakened. This lethargic diplopia is not paralytic but an exaggerated physiologic phenomenon due to retarded convergence of the visual lines after the position of the eyes during sleep.

Two other cases with diplopia from typical ocular palsies are reported.

C. Z.

Rothan, H. Influence of Adrenalin on Retinal Function. Klin. M. f. Augenh., 1925, Nov.-Dec., 75, p. 747, ill.

Rothan found in 100 persons, after instillation of three drops of adrenalin 1:1000, a considerable decrease of dark adaptation. Whether there was an influence on the pigment epithelium and the rods and cones as anatomically proven in the frog by Nakamura and Miyake and confirmed by Batschwarowa, is left undecided. Impeded circulation by capillary action might influence the photochemical process. A direct chemical action on the sensitive epithelia seems probable.

As a practical application of these investigations, adrenalin instillations, upon suggestion of Prof. Schnaudigel, removed for several hours distressing entoptic phenomena in diseases of the retina associated with irritation of the pigment epithelium, cones and rods, as in detachment of the retina, chorio- and neuroretinitis.

C. Z.

di Marzio. Primary Posttraumatic Glaucoma. Saggi di Oftalmologia, p. 31.

The author classifies the cases reported in the literature as follows: 1. Those with a wound of the eyeball, usually perforating. These are easily explained as due to the mechanical effect of incarcerated iris or a dislocated lens. 2. Cases due to lesions of the orbit, usually with fracture of the bones of the orbit, aneurism into the cavernous sinus, etc., and in this series the effect of a severe disturbance of the circulation is evidently the cause of the glaucoma. 3. Cases of contusion in which no lesion of the eyeball is produced. It is in this class of cases that the author is especially interested. The three cases which have been reported as due to a severe general contusion such as a fall in which the eye itself was not directly touched, the author considers must be doubtful. There are too few cases to establish a direct causative relation between such traumatism and glaucoma and also in these cases the severe pyschic disturbance always present may in itself be sufficient to cause glaucoma. Of the cases in which the globe itself was contused but not

wounded, the author has collected forty-four. In 12 of these, hemorrhages were present in the eye, which might be a factor in causing the glaucoma. Twenty-two were uncertain in one or more respects as to the causative relation of the trauma to the glaucoma. Only ten cases were found such as the author's own cases, in which a pure glaucoma resembling ordinary primary glaucoma occurred after contusion of the globe. The author's first patient, a man of 42, had complained of pain and the appearance of rings around the lights for several years. He first presented himself five days after he received a severe blow by a fist on the face causing a fracture of the floor of the right orbit. The right eye showed acute glaucoma with a tension of 62. The fundus showed only slight stasis but the field was distinctly limited. A trephining was performed which reduced the tension to 18 with normal central vision. Three years after the operation the patient was seen again. At this time the right or operated eye was normal while the left eye which had received no traumatism showed a tension of 40 and the symptoms of a slowly progressive simple glaucoma. His second patient was a man of 35 who received a kick from a horse in the region of the left orbit. No fracture could be demonstrated by the roentgen ray. Pain in the left eye followed immediately and when seen by the author the tension was 45 with vision of 5/10. The veins were slightly dilated. An iridectomy performed two days later resulted in normal tension with a vision of 10/10. This condition had been maintained when the patient was seen again three years later but the previously normal eye had developed acute glaucoma with a tension of 60 and vision 3/10. Iridectomy was performed with success on this eye.

In a series of ten cases of direct traumatism to the globe in which Magitot recorded the tension, a transitory hypertension was found in all, which went on to severe glaucoma in one of the ten cases. Cange was able to produce hypertension experimentally in rabbits by contusion of the globe. In the author's first case, glaucoma was, undoubtedly, already present as shown by the history and the contraction of the field. The accident simply called attention to it and possible aggravated it. In the second case, however, the trauma undoubtedly precipitated the glaucoma. The fact that the eye was predisposed to glaucoma, however, in this and also in the first case, is shown by the occurrence of glaucoma in the second untraumatized eye three years after the original injury. Thus in the second case it is quite probable that glaucoma would have occurred at some time without the occurrence of the injury. S. R. G.

Tron, E. Some Peculiarities of Astigmatism After Cataract Extraction and The Causes of Normal Curvature of the Cornea. Klin M. f. Augenh., 1925, Sept.-Oct., 75, p. 333.

From his observations with the ophthalmometer Tron found the postoperative astigmatism after cataract extraction to be distinguished from ordinary regular astigmatism by irregular decrease of refraction from one chief meridian to another. The incision produces a greater flattening of the peripheral parts of the cornea which may be explained by the different elastic properties of the sclera and cornea. The curvature of the cornea is due to the cooperation of intraocular pressure and the different elasticity of sclera and cornea. This assumption explains the greater flattening of the vertical meridian. The smaller radius of the posterior surface of the cornea than that of the anterior is understood from the effect of the intraocular pressure. C. Z.

Wodak, E. and Herrmann, G. Influence of Vestibular Excitation on Ocular Palsies. Klin. M. f. Augenh., 1925, Sept.-Oct. 75, p. 322.

In a man, aged 38, with paresis of the right abducens, a caloric horizontal rotatory nystagmus was produced to the side of the paretic abducens and the patient looked towards this side, typical nystagmus was obtained in the healthy eye, while in the paretic eye a much finer oscillating vertical nystagmus was noticed, the fast and slow components of which could not be defin-

itely distinguished. Such observations may, perhaps, be of diagnostic value in ocular palsies.

C. Z.

Fuchs, A. Neuritis Papulosa, a Rare Luetic Eye Disease. Zeitsch. f. Augenheilk., 1926, Band 59, p. 213.

The author reports the histories and ocular findings in detail of four cases, coming under his own observation, showing what he has termed neuritis papulosa. In addition he gives in outline the essentials of six similar cases taken from the literature. Characteristic symptoms and signs of this disorder are that it is unilateral and most frequently occurs in the first two years after the luetic infection. The vision becomes diminished to counting fingers or motion of the hand. The vitreous is intensely cloudy without any marked signs of inflammation being present in the anterior segment of the globe; anterior to the papilla there lies an indistinctly outlined white mass of exudate. There take place many hemorrhages in the retina, frequently recurring. After a proctracted treatment the vitreous slowly clears and the exudate shrinks up to a white cord lying upon the papilla; this cord usually extends from the papilla to a peripherally lying area of choroidal atrophy. The prognosis is usually good; a useful amount of vision is restored as a rule.

H. G. L.

Janson, E. Genesis of Glass Membranes in Chronic Inflamed Eyes. Klin. M. f. Augenh., 1925, Nov-Dec., 75, p. 681, ill.

The right eye of a boy, aged 19, was injured by an explosion seven years previously and grew totally blind. It was painful and there was increased tension. The whole interior, as far as visible, seemed to be filled with liquid, in which light gray and white slightly glistening dust granules floated about, like snow. The eye was enucleated and the histologic findings are described in detail. Large, round, pale cells, hematogenous round cells, pointed remnants of cholesterin crystals and vacuoles were found.

Glass membranes in eyes with old inflammations seem to be the result of a very gradual secretory process of the endothelia.

C. Z.

di Marzio. Cylindroma of the Lacrimal Gland. Saggi di Oftalmologia, 1924, p. 43.

The different opinions as to the proper classification of lacrimal gland tumors are discussed and twenty-three cases, all of those which the author was able to find in the literature in which the diagnosis of cylindroma could be made with fair certainty, are abstracted. Four clinical cases are presented and sections of two other cases reported, in which the operation had been performed at the ophthalmological clinic in Rome. The first case was a woman of thirty-five who had showed an exophthalmos of the right eye for the last three months with diplopia. A tumor could be felt in the region of the lacrimal gland. Motility of the eye was slightly limited in this direction, vision was 2/10 and the field was slightly limited in the direction corresponding to the tumor. The tumor was removed by a brow incision with resection of the outer wall of the orbit. It proved to be an encapsulated tumor 30 by 31 mm. in dimensions. On section the lacrimal gland itself was found to be normal and the tumor to arise from the posterior capsule of the gland. It contained cartilage, myxomatous tissue and the masses of cells enclosing a central hyaline body which have been described as characteristic of cylindroma. It apparently orginated from the endothelium of the posterior capsule, and may be called a cylindroma of the type of myxo-chronic-endothelioma. In the second case, exophthalmos had existed for several years so that the left eye was pushed eleven millimeters forward and down so that the border of the upper lid was on a level with that of the right lower lid. Motion was almost entirely absent. Vision was 2/10. The tumor was removed without any resection of the bony orbit. There has been no recurrence in eleven years. Sections showed the tumor to arise from the gland itself, and to be completely encapsulated The capsule contained principally

masses of typical cylindroma cells containing the characteristic hyaline body in the center. From the collection of the University of Rome, two other tumors were found and sections of these are reported. The tumors were similar to the second case and were considered typical cylindromata. These four cases were the only ones of lacrimal gland tumor out of 31 orbital tumors operated upon during fifteen years at the clinic at Rome. From examinations of his sections and from the other reports in the literature, the author concludes that they originate from the endothelium. They are slow growing and benign in nature. The recurrences which have been reported are probably due to incomplete removal of the tumor. S. R. G.

di Marzio. Corneal Lesions from Roentgen Ray and Ammonia. Saggi di Oftalmologia, 1924, p. 63.

Before Chalupecky had called attention to the similarity between these two types of burns when produced experimentally, the author had noticed the great clinical resemblance between them and in the present article illustrates this similarity by three cases. The first case was treated for a carcinoma at the inner angle of the lid. Secondary rays were employed, a zinc and aluminum filter being used and the eye being protected by a lead plate. Iontophoresis with a solution of mercury succinate was also employed after the irradiation. No effect on the eye was noted until one month after the treatment when an edema of the lids developed with a feeling of discomfort in the right eye. After three days, a purulent secretion appeared and a thick adherent membrane was present in both folds. The bulbar conjunctiva became a deep blue color and was adherent to the globe. A small corneal infiltrate developed and the iris showed a marked reaction. Six days later the cornea acquired a general cloudiness and the conjunctiva around the limbus became necrotic. The condition of the conjunctiva improved and the secretion disappeared but the iritis became worse, a rise of tension developed, and the corneal epithelium desquamated. Two months after the first ocular symptoms were noticed, the cornea was practically necrotic, had perforated, and the anterior chamber was empty. The eye was eviscerated. A similar dose was applied to the frontal region and directly in the pharnyx. Immediately after the second treatment a marked hyperemia of the conjunctiva and lid borders developed with a very marked purulent secretion and loss of the epithelium of the lower half of the cornea. In spite of this a third treatment was given the following day. Both corneas became entirely anesthetic and opaque. An infiltrate of the right cornea developed and then of the left. The right cornea was completely lost and the globe shrunk. Two paracenteses were done on the left cornea and this apparently was of value in preventing a similar course. Healing of the large loss of substance in this cornea proceeded very slowly, not being complete after four months. The patient died about three months after this last observation.

The third case received a splash of 33% ammonia in the right eye. One week later when first seen the bulbar conjunctiva showed necrosis very similar to that seen in the first case with loss of epithelium of the lower inner quadrant of the cornea. The whole cornea was anesthetic. The corneal erosin healed very slowly and several times when almost healed over would become detached again. Healing was not complete until after four months and vision was reduced to 5/100 by the corneal opacities. Various opinions as to the dose of roentgen ray which can be given with safety to the ocular structures are discussed. A conservative opinion is that of Jacoby who noticed damage to the cornea following twice the skin erythema dose, the lesion following a latent period of three to five weeks. The cilia were lost two to four weeks after a similar dose. One skin erythema dose produced no harm in Jacoby's experience, even when this was used several times. The author esti-

mates that his first case received two skin erythema doses of secondary rays and his second case also two skin erythema doses at each aplication, the applications being given so close together that damage to the ocular structures might have been expected. He believes that one skin erythema dose is fairly safe. The similarity of roentgen ray burns which he observed to those caused by ammonia, he believes is evidence that the damage done by the roentgen ray to the cornea is a purely chemical injury and not as some other authors have supposed, a neurotrophic change due to lesions of the corneal nerves. S. R. G.

Rosenstein, A. Maria. Rapid Growth of a Limbus Sarcoma in an Hereditary Nevus During a Koch-Weeks Conjunctivitis. Klin M. f. Augenh., 1925, Nov.-Dec., 75, p. 679.

An hereditary nevus of the conjunctiva near the limbus of a woman, aged 53, had remained stationary until during a Koch-Weeks conjunctivitis, it was converted into a malignant sarcoma and grew rapidly. As it was adherent, the extirpation was difficult. Its side was curetted and cauterized and the conjunctiva sewed. It healed and the condition was good after one and one-half years.

C. Z.

Scheel, E. Glioma Retinae in a Girl, Aged 12. Klin. M. f. Augenh., 1925. Nov.-Dec., 75, p. 670.

The histologic examination is described in detail. The tumor was a typical glioma, consisting of numerous polygonal cells, arranged in groups of about 20, radially around the vessels. It was limited to the retina in connection with both granular layers. Peculiarities were the great abundance of vessels and the leucocytes freely distributed in foci. There was no recurrence after ten months.

C. Z.

di Marzio. Angioid Streaks of the Retina. Saggi di Oftalmologia, 1924, p. 79.

Thirty cases of this condition have been reported in the literature, some of these, however, are probably not typical cases. Only two cases have been examined anatomically and in one of these the clinical history was not included. The author's case was a man of 36 who had received a blow in the right orbital region ten years before and had noticed a diminution of vision following this accident. When seen by the author, vision was 5/10 in the right eye, a relative central scotoma was present, and an absolute para-central scotoma. The retina was edematous and yellowish red in the central region. A small hemorrhage was noted near the macula and coming from this hemorrhage was a small streak of the same color which gradually became grayish blue in color. A network of similar grayish blue streaks was seen in the retina all around the nervehead, these streaks becoming smaller as they approached the equator. They were all located beneath the retinal vessels and varied greatly in size. With the slit lamp and contact glass it could be seen that these were located much deeper than the network of lymphatics which could also be distinctly made out. A network of similar lines was seen in the other eye but these were more delicate and no hemorrhages were seen. A discussion of various opinions as to the origin of these streaks is given and the author concludes that they probably all originate from retinal hemorrhages usually secondary to inflammatory changes in the choroid and retina. He agrees with Treacher Collins that the pigment seen is probably collected in the perivascular lymph spaces of the vessels in the circle of Zinn and is probably hematoidin or other blood pigment absorbed from the area of hemorrhage. Two colored plates are given to demonstrate the fundus condition.

S. R. G.

NEWS ITEMS

Personals and items of interest should be sent to Dr. Melville Black, 424 Metropolitan Building, Denver, Colorado. They should be sent in by the 25th of the month. The following gentlemen have consented to supply news from their respective sections: Dr. H. Alexander Brown, San Francisco; Dr. Wm. Thornwall Davis, Washington; Dr. Gaylord C. Hall, Louisville, Ky.; Dr. George F. Keiper, LaFayette, Indiana; Dr. J. W. Kimberlin, Kansas City, Mo.; Dr. George H. Kress, Los Angeles; Dr. Edward D. LeCompte, Salt Lake City; Dr. W. H. Lowell, Boston; Dr. G. Oram Ring, Philadelphia; Dr. Charles P. Small, Chicago; Dr. G. McD. VanPoole, Honolulu.

DEATHS.

Dr. Nelson L. North, Brooklyn, N. Y., aged sixty-one, died July 13.

Dr. Robert G. Reese of New York City died October 18, 1926.

Dr. H. H. Stark of El Paso, Texas, died October 22, 1926.

Dr. Ancil Martin, Phoenix, Ariz., aged sixty-five, died August 4th of acute nephritis.

Dr. John Lewis Washburn, Youngstown, Ohio, aged fifty-one, died June 21st of acute nephritis and rheumatic fever.

Dr. Richard Henry Lewis, Raleigh, N. C., aged seventy-six, died August 6th.

Dr. William Freeman Southard, San Francisco, aged eighty-eight, died August 8th of cerebral embolus.

SOCIETIES.

The eye and ear section of the Los Angeles County Medical Association gave a dinner, August 16th, in honor of Dr. Samuel J. Kopetzky, New York.

The Chicago Ophthalmological Society met October 18 at the Hotel Sherman. A dinner was given in honor of Dr. Nelson M. Black, Milwaukee, preceding the program. Dr. Black and Dr. Ferdinand H. Haessler addressed the society on "Dystrophic Intracellular Opacity of Corneal Epithelium."

Dr. Leslie L. McCoy, Seattle, addressed the Puget Sound Academy of Ophthalmology and Otolargngology, September 21, on "Preoperative and Postoperative Treatment of Cataract." Dr. McCoy also presented a case of cholesterin crystals in the vitreous. Dr. Morrill J. Morris presented a case of sarcoma of the choroid which, tho present about six months, was still intraocular.

The section on Ophthalmology of the College of Physicians of Philadelphia, met Thursday, October 21, 1926, at 8:00 P. M.

Program: Dr. G. E. de Schweinitz and Perce DeLong: "A Series of Microscopic Slides Illustrating Various Corneal Lesions. Lantern Demonstration." Dr. Wm. Zentmayer: "A Case showing the Visual Result Obtained by Operation for Congenital Dislocated Lens." Dr. Thos. B. Holloway: "Unusual Lens Changes in a Case of Diabetes." Dr. Burton Chance: "Presentation of Instruments used by the late Dr. William F. Norris." Dr. Warren Reese: "A Case of Posterior Lenticonus." Dr. Thos. J. Clemens: "A New Tucking Operation for the Correction of Strabismus, with Presentation of Cases." Dr. Leighton F. Appleman: "A Case of Calcareous Film of the Cornea, showing the Results of Operation."

A Smoker at the Rittenhouse Hotel in honor of the Members of the Council of the American Ophthalmological Society, was held immediately following the meeting of the Section.

The program of the All Russian Ophthalmologic Congress, which was held in Moscow, September 27th to 30th was as follows: 1 Organization of Medical Help in Eye Diseases; Measures of Fighting and Preventing Blindness. 2. Trachoma and its Prevention. 3. Chronic Eye Diseases: Methods of Diagnosis and Scientific Investigations. 4. Individual Scientific Communications.

PERSONALS.

Dr. Donald H. O'Rourke, Denver, is in Vienna for a special course.

Dr. Thos. D. Allen, Chicago, left for Vienna in September. He is planning to work there for the next six months.

Dr. Ben Witt Key announces the removal of his office to 100 West 59th Street, New York City.

The honorary degree of Doctor of Science was conferred in June upon Dr. Oscar Wilkinson, of Washington, D. C., by the Southwestern University of Memphis, Tennessee.

Mr. W. Clark Souter has been appointed Ophthalmic Surgeon to the Aberdeen Royal Infirmary.

Dr. Nora M. Fairchild, Omaha, and Dr. Beulah Cushman, Chicago, are spending the fall and winter in Europe on a study trip. They are taking special work on the slit lamp at Basel under Dr. Franceschetti, and are registered for the Fuchs course in Vienna.

The following appointments have been made at St. George's Hospital, London; Mr. Harold Grimsdale, Consulting Ophthalmic Surgeon; Mr. R. R. James, Ophthalmic Surgeon; and Mr. W. Stewart Duke-Elder, Assistant Ophthalmic Surgeon.

Col. C. L. Lynch, U. S. A. Med. Corps, Ret. has been appointed Medical Director of the Episcopal Eye, Ear and Throat Hospital, Washington, D. C.

Drs. Edw. L. Morrison, John W. Burke, J. Beaty Griffith and Wm. T. Davis have been elected members of the Senior Staff of the Episcopal Eye, Ear and Throat Hospital, Washington, D. C.

Dr. Abram B. Bruner has been appointed senior clinical instructor in ophthalmology for two years at Western Reserve University Medical School, Cleveland, Ohio.

Dr. Edward V. L. Brown has been appointed clinical professor, department of ophthalmology, Rush Medical College, Chicago.

Dr. and Mrs. Casey A. Wood, after spending the summer on a house boat at Srinagar, India, are on their way to Manila, by the way of China and Japan. They expect to return to this country some time next spring.

The board of trustees of the University of Chicago recently announced the following promotions in the department of ophthalmology, Rush Medical College: Drs. Thomas D. Allen and Earl B. Fowler, assistant clinical professors; Drs. Herman P. Davidson and James P. Fitzgerald, clinical instructors; and Drs. Richard C. Gamble, Vernon M. Leech, Aristoph Spare and Alfred L. van Dellen, clinical associates.

Dr. L. Webster Fox of Philadelphia, spent the summer in trachoma eradication and educational work among the Blackfeet Indians. He held a clinic at Browning, Montana, August 10th, which was attended by twenty-eight physicians of the Indian Service. He has been making his headquarters at Glacier National Park.

MISCELLANEOUS.

Under the will of Miss Ida Seasongood the New York Association for the Blind received $1,000.

The Maine Institute for the Blind received $2.000 by the will of the late Mrs. Elizabeth Pullen.

It has been announced that the Pittsburgh Eye and Ear Hospital, located at 1945 Fifth Avenue, will join in the medical center and erect a $1,000,000 structure opposite the new Children's Hospital on the University's site.

The Memphis Eye, Ear, Nose and Throat Hospital was opened to patients August second. There are thirty-seven rooms for private patients, and a total of seventy-two beds and five operating rooms. Twenty-five beds have been endowed as memorials. The dispensary will be opened daily for indigent patients. The Memphis Medical Journal says that the hospital is the finest of its kind in the South and that the credit for its conception, promotion and erection is due Dr. Louis Levy. There is a staff of ten surgeons in the eye department and fourteen in the ear, nose and throat department.

Current Literature

These are the titles of papers bearing on ophthalmology. They are given in English, some modified to indicate more clearly their subjects. They are grouped under appropriate heads, and in each group arranged alphabetically, usually by the author's name in *heavy-faced type*. The abbreviations means: (Ill.) illustrated; (Pl.) plates; (Col. Pl.) colored plates. Abst. shows it is an abstract of the original article. (Bibl.) means bibliography and (Dis.) discussion published with a paper.

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